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## The Massachusetts Medical Society.

## THE SHATTUCK LECTURE.*

## EPIDEMIOLOGY AND ETIOLOGY OF INFLUENZA.


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THE paucity of facts, especially of etiological facts, might stamp influenza as an unwise selection as a subject for this lecture. It is true that doctors prefer dogma to conjecture, and definite procedure to advice on general principles, but few dogmatic statements can be made concerning etiology, prophylaxis or treatment of influenza.

While it cannot be proven there is every reason for believing that influenza is a disease of great antiquity. From a period of four centuries before the Christian era accounts are found of a plague or epidemic which may have been influenza. The description of these earlier epidemics is too vague to warrant any positive conclusion.

From the sixth to the tenth century numerous epidemics are recorded with history of cough and catarrhal symptoms which are more suggestive of influenza. Hirsch[1] in his tabulation of influenza epidemics or pandemics excludes those prior to 1173 as too indefinite and uncertain to serve any useful purpose in his compilation.

There is not only uncertainty as to the nature of the disease but obviously the tabulation must be incomplete, as many epidemics occurred of which we have no record. Thus, many of the intervals shown in Hirsch's table may not have been quiescent periods but simply evidenced failure to record epidemics. No safe deductions as to cycles or periodicity can be made in these years from the 12th to the 18th century. Noah Webster[2] describes the first American epidemic in 1647 and compiles a table of epidemics from 1174 to 1797. Webster coupled the epidemics in each instance with a volcanic eruption, earthquake or some unusual climatic condition.

Webster made many interesting comments on the epidemiology of influenza. He noted that the epidemics were sometimes limited to the American continent and that contrary to usual custom in certain pandemic years, the disease beginning in America spread to the entire world. In 1698, 1757, 1761 and 1781, "it spread over the American hemisphere one year prior to its pervading the other hemisphere." In regard to incomplete records, Webster says:

"I regret my want of materials to complete

* Delivered before the Massachusetts Medical Society, June 8, 1920.

a view of this subject. No regular register has been kept in America, of the seasons, diseases and phenomena, from the first settlement, and whether any notices of all the catarrhs in this country are in existence, I do not know. I have found no accounts of any, between 1655 and 1698—nor between the latter year and 1733. One of these instances, that in 1698, came to my knowledge by accident, as I have mentioned under that year, in the foregoing history. From the uniform appearance of this epidemic as often as once in ten or twelve years, in other periods, we have ground to believe, it has always occurred in nearly the same periods."

As to the date of the first authentic epidemic of influenza there is much difference of opinion.[3] Hirsch places it as 1173; Webster 1174; Zerviani 1239; Gluge 1323; Schmeich, Haeser and others 1837; Thompson, Zulzer and Seifert 1510. Since 1510 detailed descriptions are more often available and little doubt exists as to the identity of the first real pandemic in 1580 and the pandemic in the Western Hemisphere 1647, described by Webster. In the 18th century besides rather widespread epidemics in 1709-12, there were decided pandemics in 1729, 1732, 1742, 1757-8, 1761-2 1767, 1781-2, 1788-90 and 1799.

In the 19th century because of a greater availability of detailed information we are on firmer ground and we begin to find clear pictures of pandemics very similar to those known to the present generation.

In 1824-25-26 influenza was widely epidemic in the Western Hemisphere, and in 1827 was generally diffused in Siberia and Eastern Russia.

According to Hirsch, after three years quiescence in 1830-31 one of the really great pandemics spread over the entire world. Its course was chronologically China, Philippines, Polynesia, Borneo, Sumatra, Russia, Baltic Provinces, Poland, Germany, Austria, Finland, Denmark, Belgium, France, Sweden, England, Scotland, Switzerland, Italy, Spain, and, in January, 1832, North America.

After a quiescent period of one year this pandemic was repeated. Beginning in Russia in January, 1833, it followed a course from east to west almost identical with that of 1830-31.

Three years later, in the autumn of 1836, a pandemic spread over Australia, South Africa and the East Indies—and beginning in Russia in December spread all over Europe and was reported in Mexico in July, 1837. In 1841-2 influenza was epidemic in Germany, Austria-Hungary, Ireland, Belgium, England and France. In 1843 there was a widespread epidemic in the United States and in 1844 Germany, France, and Switzerland and Russia were stricken in the order named. This seemed to be a reversal of the usual geographic progression from east to west.

Apparently, according to Hirsch, there was pandemic prevalence in 1846-8, 1850-1, 1857-8, 1874-5. The same writer states that pandemics occurred exclusively in the Western Hemisphere in 1843 and 1873.

It is true that we have more accurate knowledge of influenza during the 19th century than at any previous period, nevertheless, for practical purposes the epidemiologist must not begin his study earlier than the great pandemic of 1889-90. It is not possible to compare recent epidemics with those occurring prior to 1889 except in the most general way.

In every phase of the subject the student finds not definite precise information but rather confusion and conflict in statement and in laboratory findings as well.

It is discouraging to the average reader and especially so to the health officer who needs and seeks instruments and agencies for combating the disease. However discouraging our present lack of knowledge, the magnitude of the problem and the certainty of its return warrant the closest study of such facts as are known, and a redoubled effort in research to clear up the etiologic points now in dispute.

I have selected the subject of influenza chiefly because of its tremendous potentiality for disaster on a large scale and the certainty that future visitations may be expected. It seems wise to review such facts and observations as we possess in the hope that an exposé of our utter helplessness may stimulate further research and give us weapons with which to fight this scourge.

As a demonstration of what did happen and of what may be expected to happen again we need consider only the appalling disaster of 1918.

Never in the history of influenza has such a death toll been exacted. It is probable that in the whole history of the world no parallel will be found for the tremendous catastrophe

of 1918, if we consider the short space of time and the wide area of distribution in which the results were manifested. Statistics can never be accurate in such times of stress and world-wide estimates are notoriously inaccurate, but the data indicate that in four months a half million lives were sacrificed in the United States and that in the entire world this particular pandemic was responsible for not less than six million deaths.

At best our statistical data are far from ideal. We would prefer to have morbidity to mortality statistics, but they are so incomplete that they are practically useless. Many objections can be made against the use of mortality statistics but the fact remains that with proper corrections they afford us the best indices available for large areas.

The use of influenza mortality statistics alone is misleading because of the fact that the majority of deaths are not recorded as influenza. The combination of influenza and pneumonia deaths makes a much better index of the extent of damage done by influenza, provided we express this in terms of excess death rates, that is, the rate over and above the normal expectancy of mortality for the given period.

### WAS THE INFLUENZA PANDEMIC OF SEPTEMBER-OCTOBER, 1918, DUE TO THE SAME CAUSE AS PREVIOUS EPIDEMICS?

That the influenza of 1918 is the same disease as described by authors in ancient and medieval times is incapable of proof although it is probable from the meager and indefinite description that many of the outbreaks so described were influenza. As to the identity of the 1918 outbreak with that of 1889-90 there is more definite evidence and the similarity, epidemiologically and clinically, is so striking that we must conclude that they were due to the same cause.

The epidemiologic similarity in the two pandemics is well shown by Frost,[4] and he considers that in the history of influenza we have cycles in which great pandemics alternate with periods of relative quiescence, the length of the interval between pandemics being usually a matter of decades.

Usually preceding a real world-wide pandemic there are prepandemic increases in prevalence, amounting to considerable epidemics which have often passed unnoticed except in retrospect. The significance of these prepandemic waves is not clear although there is a natural tendency to connect them with the great pandemic rise which follows.

The special characteristics of the great pandemics are rapid spread, wide area of distribution and definite geographical progression along the most widely used routes of travel and trade. Such characteristics undoubtedly marked many of the historical epidemics tabulated by Hirsch and others, but are especially noteworthy in the pandemics of 1889-90 and 1918. These striking characteristics are lacking in the epidemics occurring in the interpandemic periods.

After a great pandemic such as '89-90 or 1918 there has been apparently such a thorough seeding of the population with the microbic cause that for years after outbreaks may occur anywhere whose spread is limited, and if the distribution is wide this seems more the development from many foci rather than an orderly geographic progression from a single source.

These post-pandemic outbreaks in succeeding years become more local and sporadic and bearing progressively less resemblance to the mode, rapidity and scope of spread of the real pandemic outbreaks.

On clinical grounds the similarity of the two great pandemics is no less probable. Kinsella[5] very positively states:

"Without doubt, the epidemic just past is of essentially the same nature as the epidemic of 1889. Then, as in the epidemic under discussion, clinicians recognized two phases or features in the disease: first, the 'influenza' proper, which lasted from three to seven days, and the complications which were regarded as secondary and not part of the original disease, though directly due to the damages that it caused. The descriptions of the disease, in England by Robertson and Elkins, in Germany by Strumpell, and in France by Duflocq, make these points clear."

Conceding the identity of the great pandemics 1889-90 and September-October, 1918, there is still doubt expressed by some as to their relationship to the relatively mild interpandemic outbreaks.

The catastrophe of September-October, 1918, is so sharply differentiated from ordinary epidemics of influenza that some have suggested that it is due to an entirely different cause. They have not gone so far as to assume that

this etiologic factor was not responsible for some previous epidemics of so-called influenza but are slow to believe that the etiologic factor responsible for the comparatively mild outbreaks is the same as that which caused the pandemic of September-October, 1918. Kinsella says:

"Perhaps the simplest and most obvious inference that can be drawn from a consideration of the epidemic of influenza that has just passed through this country is that it was something unusual, something that had not been seen for many years, and something that has departed leaving few representative cases that can ever be regarded as typical instances of the disease. Whatever the cause of the disease may be, it is clear that this agent is one to which the body is not accustomed. In fact, it would almost seem necessary to postulate at the outset that this agent is not commonly present in the body under normal circumstances, because it is difficult to conceive that any bacterium or virus that is even partially adapted to the environment of the human body should suddenly become unadapted and assume such a high degree of invasiveness as the agent of influenza possesses. Moreover, if the cause of influenza is some bacterium or virus related to a variety of bacterium or virus commonly present in normal individuals, then it would seem necessary to postulate that the cause of influenza is a very highly individualized variety."

Other writers have found difficulty in believing that the organism causing influenza in interpandemic periods, such as the mild epidemics of March, April, 1918, could so completely change as to become the cause of the frightful mortality five months later. These observers have been prone to ascribe the pandemic to virulent strains introduced from abroad.

Soper[6] inclines to the view that the organism or virus was introduced from abroad. He states that the disease was reputed to be epidemic in Spain in the early spring but that it was known to be present in the United States (Fort Oglethorpe) in March, 1918. In the latter part of March the disease appeared in the A. E. F., the French and British armies and the civil population. Many patients with influenza arrived in the United States upon ships from Europe in June, July and August, 1918. He cites many ships carrying influenza to our ports and concludes:

"The patients from the vessels were sent ashore and soon mingled with the civilian populations. There were thus scattered rather widely along the Atlantic seaboard sparks from which the pandemic not improbably arose."

Winslow and Rogers[7] suggest the same origin. From information furnished by Dr. T. E. Reeks, Connecticut State Department of Health, the following statement is made:

"Influenza first appeared as an epidemic in Connecticut in New London, in the eastern part of the State, on or about September 1, 1918, when several cases of the disease were reported by the naval hospital at New London. These cases came primarily from the experimental Station and Fort Trumbull, where vessels from foreign ports had discharged patients."

On the other hand, we must concede the possibility of an increased virulence of the strains of organisms responsible for the relatively mild prepandemic prevalence of March-April, 1918. Frost lays stress on the significant rise in general prevalence and the many definite local outbreaks in the spring of 1918 and concludes:

"The rise in mortality from this group of etiologically heterogeneous diseases in the spring of 1918 is so sudden, so marked, and so general throughout the United States as to point very clearly to the operation of a single definite and specific cause, something largely independent of meteorologic and other local conditions. The observed occurrence of local epidemics of influenza at that time in widely scattered localities, the intimate association established at Camp Funston between the epidemic of influenza and pneumonia, and the subsequent development of the influenza pandemic, all indicate that the increased pneumonia mortality of March and April, 1918, was the consequence of a beginning and largely unnoticed epidemic of influenza, and beginning, in this country, of the great pandemic which developed in the autumn."

## GENERAL EPIDEMIOLOGIC CHARACTERISTICS OF INFLUENZA.

*Prevalence Since 1889.* For intensive study there is available for certain localities fairly complete data from 1889 to date. For Massachusetts we have the deaths from influenza and pneumonia by months from 1887 to date. This covers what may be termed one complete cycle or the period from one great pandemic to an-

other, and it affords a basis of comparison between these two great pandemics.

Chart I shows the excess of annual death rates per 100,000 for influenza and pneumonia by months for Massachusetts from 1887 to 1919. After a quiescent period of several decades the pandemic beginning December, 1889, is manifest on the chart in three successive waves with highest point as follows: January, 1890; May, 1891, and January, 1892. That of January, 1890, being very high, that of May, 1891, considerably lower, and that of January, 1892, highest of all. As an aftermath of the great pandemic period 1889-1892, the chart shows a significant rise in epidemic prevalence in some month from January to April every year except 1898, 1902 and 1904, or in twelve of the fifteen years from 1890 to 1904. In 1905 and 1908 sharp rises are shown and an excessive prevalence from November, 1910, to February, 1911. From 1912 to 1914 the rates were probably as near normal as can be expected in the winter months. A noticeable rise took place March-April, 1915, with a higher peak in January, 1916, and lower peaks in March, 1917, and April, 1918, as if beginning a new cycle.

Chart II shows the annual excess death rates per 100,000 for forty-two large cities with an aggregate population of *about 22 million* by months from January, 1915, to August, 1918, inclusive. This chart very clearly shows the prepandemic rises in prevalence occurring each year in the winter and spring months, that culminating in the peak of April, 1918, being especially noteworthy.

There was then not only a regular prevalence in the winter and spring months every year but there was an epidemic prevalence in twenty-two out of thirty years and the longest period without epidemic prevalence was from February, 1911, to March, 1915, a period of four years. Further, beginning March, 1915, there has been an annual epidemic prevalence in the winter or spring months in 1915, 1916, 1917 and 1918, followed by the great pandemic of September-October, 1918.

## PERIODICITY.

Early writers asserted that there was a definite periodicity in epidemic appearances of influenza. Brownlee[8] deduced from a study of influenza mortality that epidemics occurred in 33-week cycles, and he forecasted an epidemic for January or February, 1920.

Stallybrass,[9] using statistics for Liverpool since 1890, confirms Brownlee's deductions as to the 33-week cycle.

Speare[10] considers mortality statistics unsuitable, because of their great fluctuation, for analysis by the periodogram method employed by Brownlee and says that the 33-week cycle has no existence in fact.

Speare divided the years into 13 periods of four weeks each and tabulated the frequency with which the observed week of maximum mortality fell within one or the other of the thirteen groups.

By this method he shows that the bulk of the maxima fall within the months of February and March. He explains Dr. Brownlee's conclusions by the fact that there is such great variation in the amplitude of the waves. A single epidemic such as October, 1918, would overshadow and practically eliminate many smaller epidemics when analyzed by the periodogram method. He concludes that the average interval is about one year.

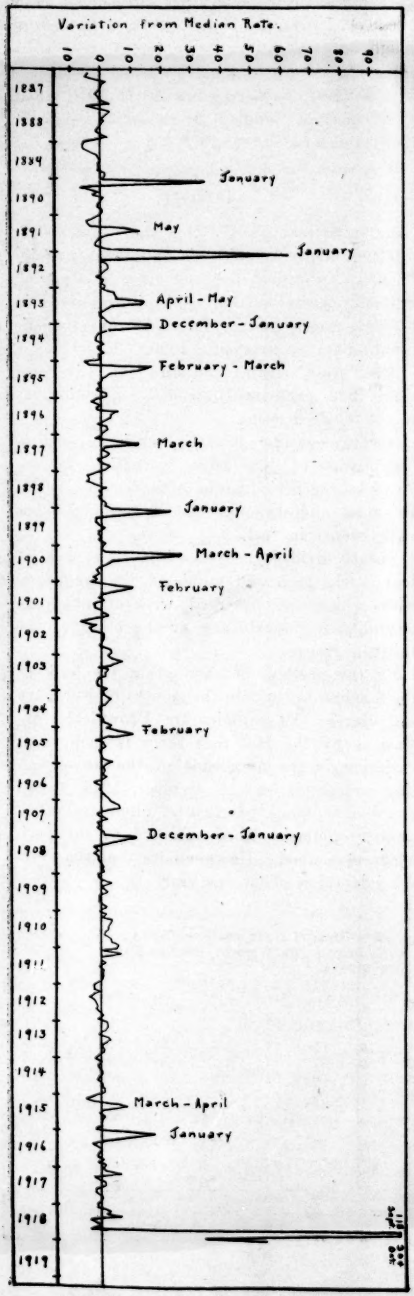
TABLE I.

ANNUAL DEATH RATE PER 100,000 AND VARIATION FROM MEDIAN RATE FOR INFLUENZA AND PNEUMONIA (ALL FORMS) FOR 42 LARGE CITIES INCLUDED IN THE WEEKLY HEALTH INDEX OF THE U. S. CENSUS BUREAU.

| | JAN. | FEB. | MAR. | APR. | MAY | JUNE | JULY | AUG. | SEPT. | OCT. | NOV. | DEC. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ANNUAL DEATH-RATE PER 100,000. | | | | | | | | | | | |
| 1915 | 231 | 264 | 296 | 284 | 151 | 114 | 76 | 67 | 77 | 106 | 153 | 354 |
| 1916 | 446 | 266 | 258 | 213 | 170 | 102 | 80 | 77 | 87 | 112 | 162 | 246 |
| 1917 | 389 | 346 | 303 | 246 | 204 | 115 | 75 | 69 | 91 | 129 | 178 | 235 |
| 1918 | 315 | 312 | 402 | 421 | 187 | 96 | 79 | 56 | | | | |
| | MEDIAN RATE (PER 100,000) FOR PERIOD 1910-1916. | | | | | | | | | | | |
| | 279 | 280 | 290 | 234 | 177 | 109 | 82 | 73 | 82 | 111 | 163 | 228 |
| | VARIATION OF ANNUAL RATE FROM MEDIAN RATE. | | | | | | | | | | | |
| 1915 | —48 | —16 | 6 | 50 | —26 | 5 | —6 | —6 | —5 | —5 | —10 | 126 |
| 1916 | 167 | —14 | —32 | —21 | —7 | —7 | —2 | 4 | 5 | 1 | —1 | 18 |
| 1917 | 110 | 66 | 13 | 12 | 27 | 6 | —7 | —4 | 9 | 18 | 15 | 7 |
| 1918 | 36 | 32 | 112 | 187 | 10 | —13 | —3 | —17 | | | | |

CHART I.

MASSACHUSETTS EXCESS DEATH RATES FROM INFLUENZA AND PNEUMONIA, BY MONTHS, 1887 TO 1918.




SEASONAL.

While no claim for annual periodicity is made, a glance at Chart I reveals the remarkable predilection which influenza has for the winter and spring months.

Every significant rise on Chart I from 1887 to 1918 occurred in the months from December to May. The pandemic of September-October, 1918, was the great and only exception to this rule.

MORTALITY STATISTICS ACCORDING TO AGE AND SEX.

In the pandemic of 1889-90 Abbott[11] states that the deaths from pneumonia and acute bronchitis for the first two weeks in 1889 and 1890 in Paris were as follows:

| AGES | FIRST TWO WEEKS 1889 | 1890 |
|---|---|---|
| 0— 4 | 45 | 111 |
| 5—19 | 6 | 22 |
| 20—39 | 7 | 127 |
| 40—59 | 14 | 249 |
| over 60 | 35 | 290 |

This table shows the excess of deaths to be chiefly in the age groups from 20 years upward. He presents other tables which show that in Paris the mortality in male adults was double that of female adults.

Parsons[12] shows that the deaths from influenza in London for the epidemic of 1847-8 and for the first quarter of 1890 occurred in the different age groups in the following percentages:

| AGES | PERCENTAGE 1847-8 | 1890 |
|---|---|---|
| under 1 | 10.5 | 5.2 |
| 1— 5 | 13.1 | 4.3 |
| 5—20 | 3.8 | 4.7 |
| 20—40 | 8.6 | 24.7 |
| 40—60 | 18.5 | 36.2 |
| 60—80 | 16.9 | 22.4 |
| above 80 | 8.6 | 2.5 |

Winslow and Rogers[13] analyzing statistics for Connecticut for the last four months of each of the years 1917-1918, show that the normal distribution of deaths in 1917 was 25% of all deaths occurred under 5 years; 25% occurred between 5 and 40 and the remaining 50% over 40. In 1918 the distribution was strikingly different; instead of 25%, between the ages of 5 and 40, this period included 49% of all deaths and 40% of all deaths occurred in the two decades between 20 to 40, as against only 17% in 1917. Considering influenza and pneumonia alone, the age period 20 to 40 in-

cluded 56% of the deaths, while only 9% occurred in ages over 49. The decade 20-29 was most severely affected, including 30% of all deaths.

The high rates of influenza mortality in certain age groups may be either due to an excessive incidence of influenza, or to a very high case fatality rate within these age groups. It is therefore necessary to study the limited morbidity survey statistics available.

CHART II.

MONTHLY VARIATION IN THE DEATH RATE FROM INFLUENZA AND PNEUMONIA FROM THAT OF CORRESPONDING MONTH IN MEDIAN YEAR, IN 42 LARGE CITIES INCLUDED IN WEEKLY HEALTH INDEX OF U. S. CENSUS BUREAU.

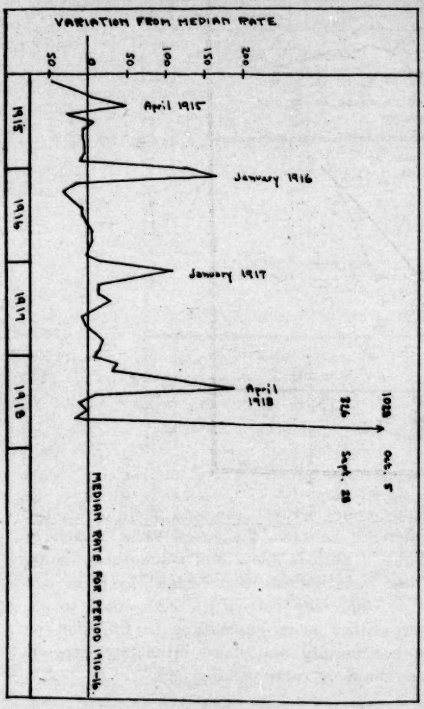



CHART III.

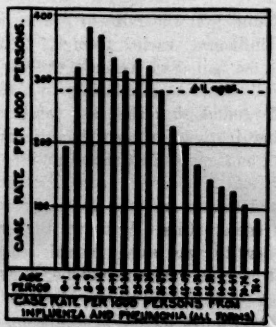



CHART IV.

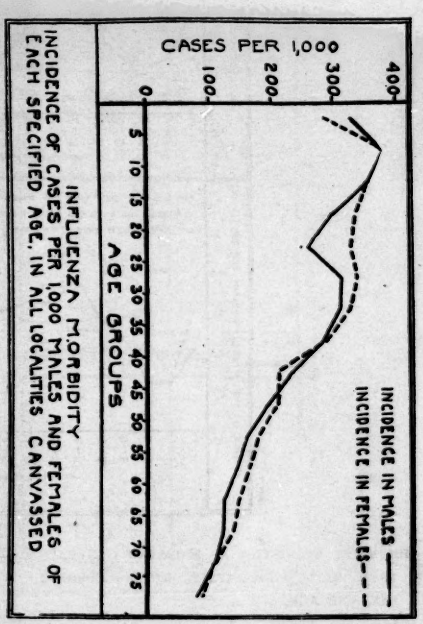



CHART V.

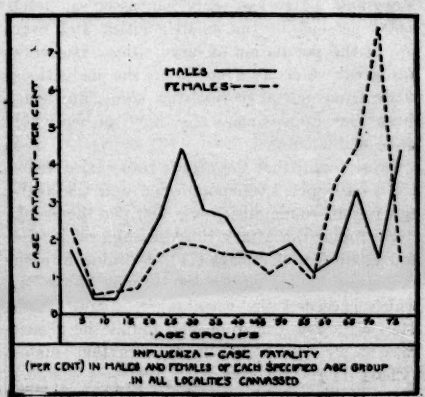
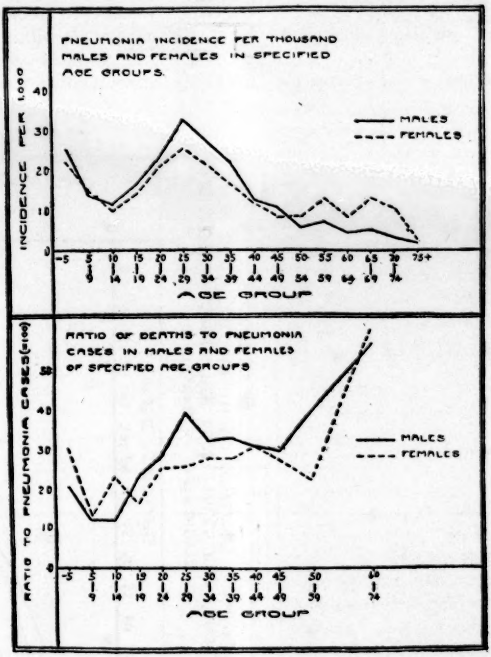


CHART VI.




MORBIDITY STATISTICS AS SHOWING GENERAL ATTACK RATE AND ATTACK RATE ACCORDING TO SEX AND AGE.

Frost[14] analyzed the results of a morbidity survey of 10 cities from 25,000 to 600,000 and in several smaller cities and rural areas in Maryland. His assistants canvassed at least 5,000 persons in the smaller cities and over 5% of the population of large cities. His work has great value as a check on the deductions made from mortality statistics alone, and confirms the approximate accuracy of many of these deductions.

Frost found that the attack rate varied from 150 to 405 per 1,000 population and the average for all communities was 280 per thousand.

He found the attack rate was highest in the age group 5 to 9 (Chart III), declining in each successive higher age group, except 25 to 34, which exceeded the rates for the ages 15 to 24. With few exceptions he found the attack rate at all ages higher in females than males (Chart IV). He says:

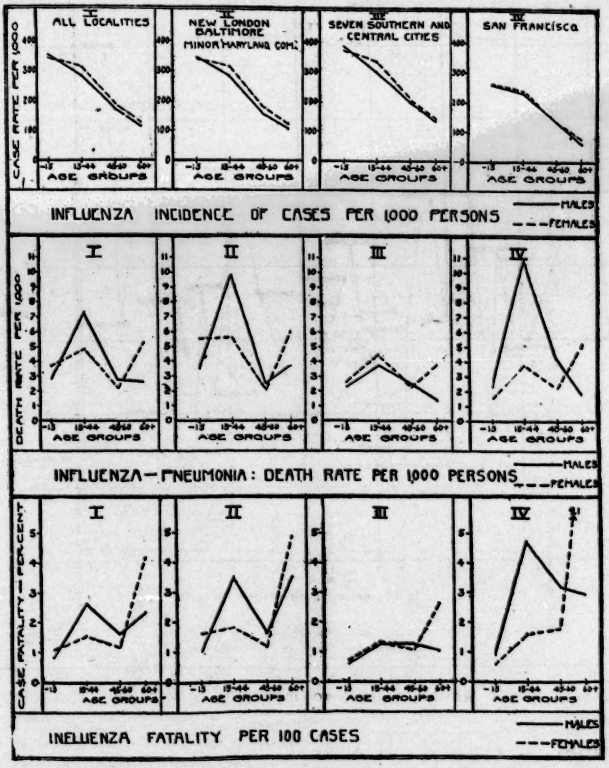
"The most striking excess of incidence in females occurs between the ages of 25 to 40, the difference between the sexes being relatively slight in periods above and below these limits. These facts indicate that females over the age of 15, especially betwen the ages of 15 to 45, were either more susceptible to infection or more generally and more intimately exposed than males of corresponding age."

CASE FATALITY RATE.

Frost found that the ratio of deaths to total cases of influenza varied from 3.1% in New London to 0.8% in San Antonio, the variations showing no consistent relation to incidence rates. He found that the case fatality rates were higher in the Northern Atlantic and Pacific coasts and lowest in Central and Southern cities.

In regard to sex and age he found remarkable differences. Chart V shows the case fatality under 15 was somewhat higher in females and over 60 very much higher in females; while from 15 to 60 there was a much higher case fatality rate in males. The lower case fa-

Chart VII.




tality rate in females from 15 to 60 seems to be explained in part at least by the lesser incidence of pneumonia in this group (Chart VI).

Winslow and Rogers, from the reports of the New Haven Visiting Nurses' Association, conclude that the case fatality rate of 4.3 per 100 cases (736 cases—32 deaths) is too high since undoubtedly many light cases failed to receive nursing care. Winslow and Rogers also analyzed information supplied by Reeks from a survey in New Britain, Connecticut, and says these figures would indicate a morbidity rate of 234 per 1,000 and a fatality rate of 3.9 deaths per 100 cases. Here again they note that light cases were incompletely reported and that this rate is probably too high. They conclude that the attack rate in Connecticut was from 200 to 400 per 1,000 and the case fatality rate from two to four deaths per 100 cases, the higher morbidity and lower fatality rate being more likely to be correct (Chart VII).

### RACE.

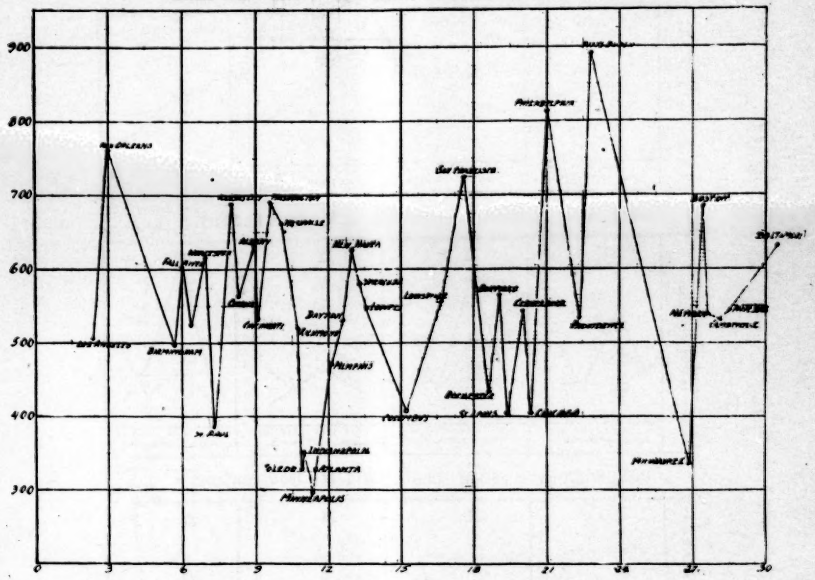
Winslow and Rogers made one striking observation in regard to race. They found that native Americans of Irish, English, or German descent had a relatively low mortality rate, while Slavic or Latin peoples had a very high mortality rate. They admit that the age distribution and economic environment account for some of the excess, but believe that these factors cannot explain the enormous differences which are shown by the Italian population.

### DENSITY OF POPULATION.

Table II shows the total annual excess death rate per 100,000 influenza and pneumonia, and

CHART VIII.

EXCESS DEATH RATE EPIDEMIC PERIOD, SEPTEMBER, 1918—JUNE 28, 1919.




Density of population—Persons per acre.

CHART IX.

VALUES FOR INDEX OF EXPLOSIVENESS.

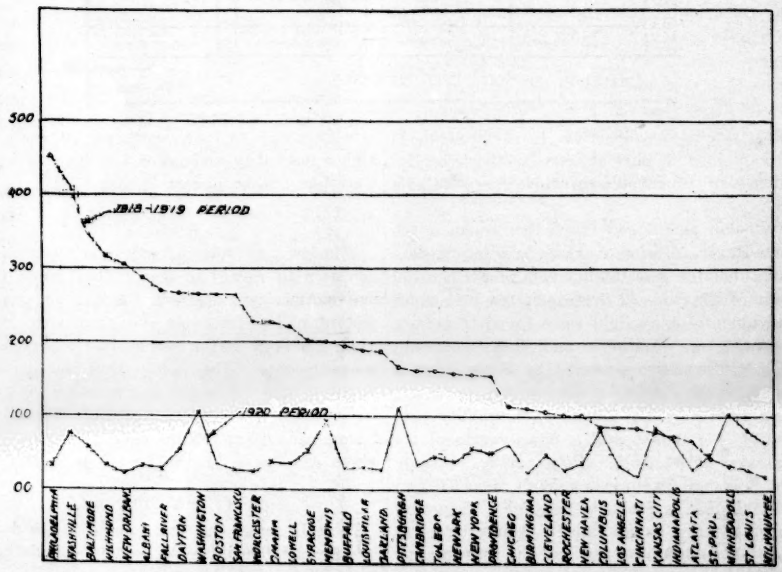


CHART X.
RELATIVE FIGURES—DEATH RATES.

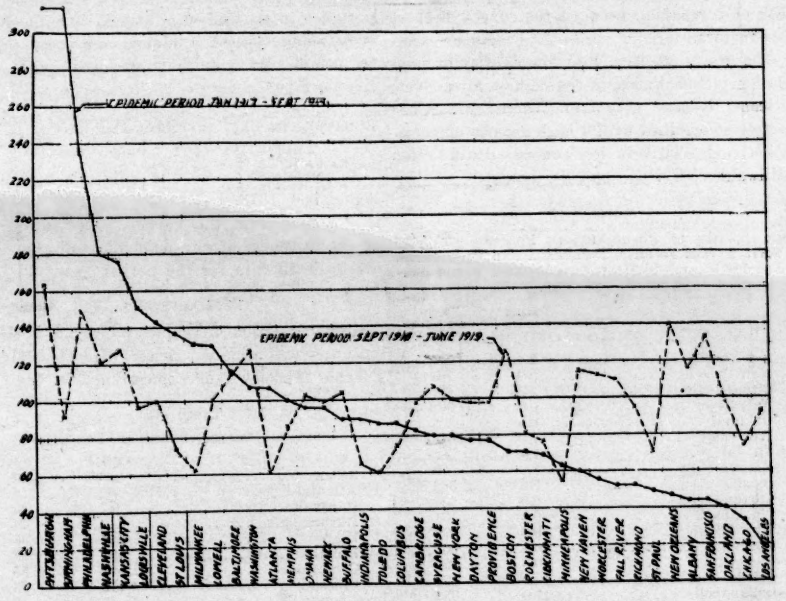
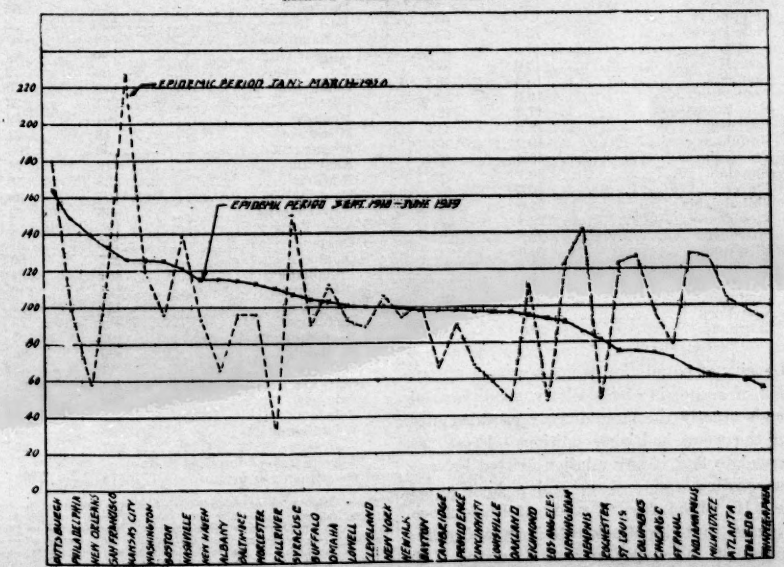



CHART XI.
RELATIVE FIGURES—DEATH RATES.



the density of population in persons per acre in 40 cities. Chart VIII shows graphically the lack of correlation between the curves of these two factors.

The group of cities with low density of population included some of the highest rates (New Orleans, Kansas City and Worcester). Conversely some cities with a high density of population had relatively low excess death rates (Milwaukee, Chicago and St. Louis).

TABLE II.

THE DENSITY OF POPULATION IN PERSONS PER ACRE WITH EXCESS DEATHS, INFLUENZA AND PNEUMONIA, ALL FORMS, PER 100,000, FOR EPIDEMIC PERIOD SEPTEMBER 8, 1918, TO JUNE 28, 1919.

| CITY | DENSITY PERSONS PER ACRE | EXCESS DEATH RATE 1918-1919 |
|---|---|---|
| Los Angeles | 2.4 | 505 |
| New Orleans | 3.0 | 761 |
| Birmingham | 5.7 | 497 |
| Fall River | 6.0 | 602 |
| Oakland | 6.4 | 528 |
| Worcester | 7.0 | 618 |
| St. Paul | 7.4 | 386 |
| Kansas City | 8.0 | 689 |
| Omaha | 8.3 | 562 |
| Albany | 8.9 | 631 |
| Cincinnati | 9.1 | 531 |
| Washington | 9.6 | 689 |
| Nashville | 10.1 | 662 |
| Richmond | 10.8 | 520 |
| Toledo | 10.9 | 329 |
| Indianapolis | 11.0 | 354 |
| Minneapolis | 11.3 | 290 |
| Atlanta | 14.4 | 330 |
| Memphis | 12.1 | 470 |
| Dayton | 12.6 | 532 |
| New Haven | 13.1 | 629 |
| Syracuse | 13.3 | 580 |
| Lowell | 13.6 | 548 |
| Columbus | 15.2 | 409 |
| Louisville | 16.6 | 530 |
| San Francisco | 17.6 | 724 |
| Rochester | 18.6 | 433 |
| Buffalo | 19.0 | 567 |
| St. Louis | 19.4 | 406 |
| Cleveland | 20.1 | 546 |
| Chicago | 20.3 | 404 |
| Philadelphia | 21.0 | 814 |
| Providence | 22.4 | 534 |
| Pittsburgh | 22.8 | 891 |
| Milwaukee | 26.9 | 337 |
| Boston | 27.4 | 685 |
| Newark | 27.5 | 538 |
| Cambridge | 28.2 | 532 |
| New York | 29.5 | 544 |
| Baltimore | 30.6 | 629 |

Density of population is not always a reliable index of economic and sanitary conditions, although usually the most densely crowded cities have the worst housing conditions. Frost[15] attempted to find if any relation existed between overcrowding and poverty and a high attack rate. He found from a study of Little Rock, Arkansas, that the attack rate increased as the number of rooms per person decreased. He says that these limited statistics suggest that domestic environment is a factor of some importance in the influenza attack rate, the tendency being toward a higher morbidity under the complex of conditions associated with relative poverty."

VARIATION IN EXPLOSIVENESS AND EXCESS DEATH RATES IN FORTY LARGE CITIES.

A consideration of the epidemic beginning September, 1918, brings out the very remarkable variation in the various cities in degree of explosiveness of the epidemic and the total excess death rate for the period.

EXPLOSIVENESS.

Great variation in explosiveness in individual cities was apparent in the 1918 epidemic.

Table III shows the explosiveness of the epi-

TABLE III.

INDEX OF EXPLOSIVENESS OF EPIDEMICS.

| CITY | 1918-19 | 1920 |
|---|---|---|
| Albany | 285 | 33 |
| Atlanta | 62 | 28 |
| Baltimore | 347 | 58 |
| Birmingham | 112 | 24 |
| Boston | 264 | 35 |
| Buffalo | 197 | 27 |
| Cambridge | 162 | 35 |
| Chicago | 115 | 63 |
| Cincinnati | 85 | 19 |
| Cleveland | 107 | 49 |
| Columbus | 87 | 84 |
| Dayton | 268 | 54 |
| Fall River | 270 | 28 |
| Indianapolis | 74 | 67 |
| Kansas City | 80 | 84 |
| Los Angeles | 86 | 32 |
| Louisvillle | 189 | 29 |
| Lowell | 221 | 36 |
| Memphis | 201 | 92 |
| Milwaukee | 18 | 64 |
| Minneapolis | 33 | 103 |
| Nashville | 416 | 76 |
| Newark | 128 | 58 |
| New Haven | 101 | 38 |
| New Orleans | 305 | 22 |
| New York | 127 | 57 |
| Oakland | 189 | 27 |
| Omaha | 227 | 38 |
| Philadelphia | 451 | 31 |
| Pittsburgh | 168 | 110 |
| Providence | 124 | 50 |
| Richmond | 314 | 29 |
| Rochester | 102 | 27 |
| St. Louis | 29 | 80 |
| St. Paul | 44 | 49 |
| San Francisco | 264 | 27 |
| Syracuse | 202 | 53 |
| Toledo | 132 | 43 |
| Washington | 266 | 104 |
| Worcester | 227 | 24 |
| All cities considered as a single population group | 78 | 33 |

NOTE.—The index of explosiveness has been computed for each city as follows: The death rate from influenza and pneumonia (all forms) in the peak week (i. e., week in which the maximum mortality occurred) has been divided by the number of weeks from the beginning of the epidemic up to and including the peak week. It must be remembered that the result is only approximate.

demic in both the 1918 and 1920 epidemics in forty cities, using as an index the death rate for the peak week divided by the number of weeks from the beginning of the epidemic up to and including the peak week. This gives an arbitrary figure which will serve as an index perhaps quite as well as that secured by more complicated methods.

Chart IX shows the great variation in explosiveness in graphic form. The cities are arranged according to the degree of explosiveness. In the 1918 epidemic eleven cities showed a very high degree of explosiveness (451 to 264) measured by this index *viz.*, Philadelphia, Nashville, Baltimore, Richmond, New Orleans, Albany, Fall River, Dayton, Washington, Boston and San Francisco. Next with high rates (227 to 189) come Worcester, Omaha, Lowell, Syracuse, Memphis, Buffalo, Louisville, and Oakland. Next comes a group with moderate explosiveness (168 to 101), Pittsburgh, Cambridge, Toledo, Newark, New York, Providence, Chicago, Birmingham, Cleveland, Rochester and New Haven.

The last ten cities may be said to have had a low index of explosiveness (87 to 18). These cities were Columbus, Los Angeles, Cincinnati, Kansas City, Indianapolis, Atlanta, St. Paul, Minneapolis, St. Louis and Milwaukee.

Geographically the distribution of the eleven highest cities was wide and included cities in New England, Middle Atlantic, Gulf and Pacific coast cities, with only Nashville and Dayton in the Middle West. The distribution in the next group of cities with high indices including New England, New York State, Oakland, California, and two cities in the Ohio or Mississippi Basin—Louisville and Memphis. The moderate group had Pittsburgh, Cleveland, Toledo and Chicago in the Middle West, Cambridge, New Haven, Providence, Newark, Rochester and New York in the East with Birmingham in the South. The ten cities with a low explosive index were distributed as follows: Los Angeles on the Pacific Coast, Atlanta in the South and Columbus, Cincinnati, Indianapolis and St. Louis in the Middle West with farther north St. Paul, Minneapolis and Milwaukee.

In general the 1920 index of explosiveness did not show such extremes of variation. There were, however, sharp rises above the average for several cities. The index of explosiveness was relatively high in 1920 in Nashville, Memphis, Washington, Pittsburgh, Columbus, Kansas City and Minneapolis. Of these Nashville, Washington and Memphis had a high explosive index in both epidemics. Pittsburgh was moderate in 1918 and high in 1920. Columbus, Kansas City and Minneapolis had a low index in 1918 and a high index in 1920.

What relation did the explosiveness bear to the actual excess death rate for the epidemic period? Of the eleven cities with highest indices for explosiveness, eight were also among the eleven cities with the highest actual excess death rates for the epidemic period. These cities were Philadelphia, Nashville, Baltimore, New Orleans, Albany, Washington, Boston and San Francisco. Thirteen cities had an actual excess death rate for the 1918 epidemic period of 600 or over, ten of these also had a very high explosive index. The three exceptions were Pittsburgh with a moderate explosive index and the highest mortality, Kansas City and New Haven with low explosive index and a very high total mortality.

Ten cities had a low explosive index of less than 100, *viz.*, Columbus, Los Angeles, Cincinnati, Kansas City, Indianapolis, Atlanta, St. Paul, Minneapolis, St. Louis, Milwaukee.

All of these had relatively low excess mortality rates for the epidemic period except Los Angeles and Cincinnati with moderate rates and Kansas City with a very high excess mortality rate.

In spite of the notable exceptions there seems to have been some correlation between the degree of explosiveness and the amount of mortality for the whole epidemic period.

What are the reasons for the great variation in explosiveness shown by the forty large cities?

Pearl[16] in an excellent monograph analyzes geographical position, density of population, differences in distribution of age groups, and great increase or decrease in population in relation to explosiveness. In regard to these factors, which have not been without effect in other diseases, he says:

"The general conclusion to which we come from an examination of the correlation data assembled to this point is that these four general demographic factors,—density of population, geographical position, age distribution of population and rate of recent growth in population have practically nothing to do, either severally or collectively with bringing about those dif-

ferences between the several cities in respect to explosiveness of the outbreak of epidemic mortality in which we are interested."

Pearl believes that the most significant factor in causing the variation to be the relative normal liability of the inhabitants to die of one of the three great causes of death,—disease of the lungs, heart, or kidneys. He says:

"Such an analysis, by the method of multiple correlation, appears to demonstrate that an important factor so far found in causing the observed wide variation amongst these 39 American cities in respect of the explosiveness of the outbreak of epidemic influenza mortality in the autumn of 1918 was the magnitude of the normal death rates observed in the same communities, particularly those death rates from pulmonary tuberculosis, diseases of the heart and of the kidneys."

Table IV shows the fifteen cities with the highest explosive index in September-October, 1918,—and the combined death rate for pneumonia, tuberculosis, heart disease, and nephritis —eleven of the fifteen cities have a very high combined death rate for these four diseases (average for 1911 to 1917 inclusive). The exceptions are Dayton, Lowell, Syracuse, and Omaha. Dayton, Lowell and Syracuse had average rates for pneumonia and heart disease, but Omaha had low rates for all four diseases.

TABLE IV.

| City | Explosive Index | Combined Death Rate per 100,000 Pneumonia, Tuberculosis, Heart Disease and Nephritis |
|---|---|---|
| Philadelphia | 451 | 726 |
| Nashville | 416 | 723 |
| Baltimore | 347 | 802 |
| Richmond | 314 | 790 |
| New Orleans | 305 | 919 |
| Albany | 285 | 835 |
| Fall River | 270 | 635 |
| Dayton | 268 | 589 |
| Washington | 266 | 765 |
| Boston | 264 | 684 |
| San Francisco | 264 | 688 |
| Worcester | 227 | 641 |
| Omaha | 227 | 469 |
| Lowell | 221 | 550 |
| Syracuse | 202 | 545 |

Of the four principal factors in our total mortality pneumonia and heart disease are the ones most likely to affect explosiveness deduced from mortality statistics. If the combined death rate per 100,000 (average for 1911 to 1917 inclusive) for pneumonia and heart disease was more than 350 the explosiveness was marked.

TABLE V.

| City | Combined Death Rate per 100,000 Pneumonia and Heart Disease | Explosive Index |
|---|---|---|
| Boston | 412 | 264 |
| Worcester | 412 | 227 |
| Pittsburgh | 403 | 168 |
| Baltimore | 393 | 347 |
| New Haven | 384 | 101 |
| New Orleans | 381 | 305 |
| Nashville | 380 | 416 |
| Washington | 376 | 266 |
| Fall River | 375 | 270 |
| Richmond | 374 | 314 |
| Philadelphia | 367 | 451 |
| San Francisco | 362 | 264 |

The twelve cities with the highest combined rates for pneumonia and heart disease (Table V) showed a very high explosive index in 1918. There are two exceptions,—Pittsburgh and New Haven; of these Pittsburgh had the highest explosive index for the 1920 epidemic, but New Haven showed a low degree of explosiveness in both epidemic periods.

### VARIATION IN SEVERITY MEASURED BY EXCESS DEATH RATES.

What are the reasons for the very great variation in the severity of the epidemic as measured by mortality in certain cities? Why did Pittsburgh have a very high rate in all three epidemic periods and why did cities like Milwaukee, St. Paul and Minneapolis have low rates in all periods?

Table VI shows in the first column what may be considered a rough index of the total damage in the forty large cities from influenza and pneumonia combined. The figure given for each city is the sum of the excess death rates for the three epidemic periods from January, 1917, to March, 1920. In the other columns are given the general death rate per 1,000 and the death rate per 100,000 for pneumonia, tuberculosis, heart disease and nephritis.

Considered as a whole there seems to be some correlation between the loss from influenza and the death rates from certain diseases. Considering the cities in three groups this correlation is even more striking.

| | Total Excess Death Rate |
|---|---|
| Group 1 | above 800 |
| Group 2 | 600 to 800 |
| Group 3 | below 600 |

TABLE VI.

| CITY | EXCESS DEATH RATE PER 100,000 FOR 3 EPIDEMIC PERIODS, JAN., 1917, TO JUNE, 1920. | GEN'L DEATH RATE PER 1,000 AVERAGE FOR 1914-1917, INCLUSIVE | DEATH RATE PER 100,000 AVERAGE FOR 1911 TO 1917, INCLUSIVE | | | |
|---|---|---|---|---|---|---|
| | | | Pneumonia | Tuberculosis | Heart Disease | Nephritis |
| Albany, N. Y. | 728 | 19.4 | 157.5 | 254.5 | 231.3 | 191.2 |
| Atlanta, Ga. | 533 | 15.7 | 167.3 | 148.0 | 112.6 | 176.4 |
| Baltimore, Md. | 810 | 18.1 | 210.3 | 233.3 | 182.9 | 175.3 |
| Birmingham, Ala. | 855 | 16.8 | 171.0 | 217.5 | 115.5 | 105.2 |
| Boston, Mass. | 822 | 16.4 | 203.3 | 173.9 | 208.5 | 98.0 |
| Buffalo, N. Y. | 722 | 15.6 | 158.2 | 156.6 | 165.3 | 118.6 |
| Cambridge, Mass. | 659 | 13.4 | 155.5 | 192.8 | 170.9 | 76.0 |
| Chicago, Ill. | 524 | 14.4 | 188.0 | 162.2 | 150.4 | 110.2 |
| Cincinnati, Ohio | 650 | 16.1 | 142.4 | 242.5 | 200.5 | 157.2 |
| Cleveland, Ohio | 740 | 14.1 | 139.9 | 144.1 | 117.8 | 92.7 |
| Columbus, Ohio | 598 | 14.9 | 130.0 | 116.8 | 151.4 | 85.6 |
| Dayton, Ohio | 690 | 14.6 | 132.7 | 157.5 | 176.4 | 102.3 |
| Fall River, Mass. | 673 | 16.6 | 219.6 | 157.4 | 155.1 | 103.0 |
| Indianapolis, Ind. | 546 | 15.6 | 133.9 | 199.4 | 177.3 | 106.3 |
| Kansas City, Mo. | 1043 | 14.9 | 137.6 | 163.7 | 138.6 | 121.3 |
| Los Angeles, Calif. | 559 | 12.5 | 90.8 | 232.4 | 161.4 | 104.3 |
| Louisville, Ky. | 701 | 15.7 | 145.9 | 198.5 | 158.0 | 150.8 |
| Lowell, Mass. | 736 | 16.1 | 161.4 | 139.3 | 156.2 | 92.7 |
| Memphis, Tenn. | 684 | 20.4 | 170.7 | 270.6 | 136.2 | 174.4 |
| Milwaukee, Wis. | 537 | 12.2 | 133.2 | 100.8 | 97.9 | 74.1 |
| Minneapolis, Minn. | 437 | 11.9 | 109.8 | 140.6 | 107.4 | 93.6 |
| Nashville, Tenn. | 933 | 17.3 | 172.4 | 229.3 | 207.9 | 112.9 |
| Newark, N. J. | 702 | 14.2 | 156.5 | 178.9 | 140.3 | 135.5 |
| New Haven, Conn. | 765 | 16.5 | 210.2 | 140.9 | 173.4 | 130.0 |
| New Orleans, La. | 854 | 20.0 | 159.2 | 283.9 | 220.7 | 255.1 |
| New York, N. Y. | 708 | 13.9 | 191.2 | 190.5 | 152.8 | 135.0 |
| Oakland, Calif. | 666 | 11.0 | 97.8 | 126.8 | 189.3 | 87.5 |
| Omaha, Neb. | 743 | 13.3 | 155.3 | 111.2 | 111.5 | 90.5 |
| Philadelphia, Pa. | 1087 | 16.2 | 166.2 | 195.1 | 191.5 | 173.0 |
| Pittsburgh, Pa. | 1305 | 16.6 | 275.1 | 135.4 | 127.7 | 86.8 |
| Providence, R. I. | 680 | 15.2 | 168.7 | 158.1 | 159.1 | 130.5 |
| Richmond, Va. | 608 | 19.2 | 179.4 | 220.9 | 194.4 | 195.1 |
| Rochester, N. Y. | 534 | 14.5 | 135.3 | 118.0 | 183.3 | 133.4 |
| St. Louis, Mo. | 629 | 14.7 | 158.1 | 154.8 | 137.7 | 167.7 |
| St. Paul, Minn. | 510 | 11.0 | 92.8 | 132.6 | 105.8 | 80.0 |
| San Francisco, Calif. | 874 | 15.5 | 128.3 | 198.6 | 234.1 | 126.6 |
| Syracuse, N. Y. | 787 | 14.6 | 141.9 | 114.7 | 178.5 | 110.0 |
| Toledo, Ohio | 431 | 16.6 | 123.3 | 189.2 | 158.9 | 91.7 |
| Washington, D. C. | 885 | 17.6 | 152.8 | 221.2 | 225.3 | 165.3 |
| Worcester, Mass. | 754 | 16.4 | 187.5 | 147.4 | 224.3 | 82.3 |

Group 1:—The cities having the highest excess death rates combined for the three epidemic periods (January, 1917, to March, 1920) also had very high rates of pneumonia, tuberculosis, heart disease and nephritis and a high general death rate. There was one exception—Kansas City.

TABLE VII.

| CITY | GENERAL DEATH RATE PER 100,000 | TOTAL EXCESS DEATH RATE, INCL. PNEUMONIA FOR 3 EPIDEMIC PERIODS—JAN., 1917, TO MARCH, 1920 | DEATH RATE PER 100,000, 1911—1917, INC. | |
|---|---|---|---|---|
| | | | Pneumonia | Pneumonia, Tuberculosis, Heart Disease and Nephritis |
| Pittsburgh | 16.6 | 1305 | 275.0 | 625.0 |
| Philadelphia | 16.2 | 1087 | 166.2 | 725.8 |
| Kansas City | 14.9 | 1043 | 137.6 | 561.2 |
| Nashville | 17.3 | 933 | 172.4 | 722.5 |
| Washington | 17.6 | 885 | 152.8 | 764.6 |
| San Francisco | 15.5 | 874 | 128.3 | 687.6 |
| Birmingham | 16.8 | 855 | 171.0 | 609.2 |
| New Orleans | 20.0 | 854 | 159.2 | 918.9 |
| Boston | 16.4 | 822 | 203.3 | 683.7 |
| Baltimore | 18.1 | 810 | 210.3 | 801.8 |
| Average for registration cities | | 717 | 158.6 | 593.6 |

Table VII shows the rates for these cities compared with the average in all registration cities.

TABLE VIII.

| CITY | GENERAL DEATH RATE PER 100,000 | TOTAL EXCESS DEATH RATE, INCL. PNEUMONIA FOR 3 EPIDEMIC PERIODS—JAN., 1917, TO MARCH, 1920 | DEATH RATE PER 100,000, 1911—1917, INC. | |
|---|---|---|---|---|
| | | | Pneumonia | Pneumonia, Tuberculosis, Heart Disease and Nephritis |
| Syracuse | 14.6 | 787 | 141.9 | 545.1 |
| New Haven | 16.5 | 765 | 210.2 | 654.5 |
| Worcester | 16.4 | 754 | 187.5 | 641.5 |
| Omaha | 13.3 | 743 | 155.3 | 468.5 |
| Cleveland | 14.1 | 740 | 139.9 | 494.5 |
| Lowell | 16.1 | 736 | 161.4 | 549.6 |
| Albany | 19.4 | 728 | 157.5 | 834.5 |
| Buffalo | 15.6 | 722 | 158.2 | 598.7 |
| New York | 13.9 | 708 | 191.2 | 669.5 |
| Newark | 14.2 | 702 | 156.5 | 611.2 |
| Louisville | 15.7 | 701 | 145.9 | 633.2 |
| Dayton | 14.6 | 690 | 132.7 | 568.9 |
| Memphis | 20.4 | 684 | 170.7 | 751.9 |
| Providence | 15.2 | 680 | 168.7 | 616.4 |
| Fall River | 16.6 | 673 | 219.6 | 635.1 |
| Oakland | 11.0 | 666 | 97.8 | 501.4 |
| Cambridge | 13.4 | 659 | 155.5 | 595.2 |
| Cincinnati | 16.1 | 650 | 142.4 | 742.6 |
| St. Louis | 14.7 | 629 | 158.1 | 618.3 |
| Richmond | 19.2 | 608 | 179.4 | 789.8 |

Group 2:—Had rather severe visitations of influenza with moderate and in some instances high rates for pneumonia, and pneumonia, tuberculosis, heart disease and nephritis combined.

Group 3:—Had very low rates for influenza with low general death rates and low rates for pneumonia, and pneumonia, tuberculosis, heart disease and nephritis combined. There were some exceptions to this rule. The general death rates in Toledo and Indianapolis are a little higher than in the other cities of the group. Chicago and Atlanta had a pneumonia rate above the average—but all the others were far above the average for pneumonia.

TABLE IX.

| CITY | GENERAL DEATH RATE PER 100,000 | TOTAL EXCESS DEATH RATE, INFLUENZA FOR 3 EPIDEMIC PERIODS—JAN., 1917, TO MARCH, 1920 | DEATH RATE PER 100,000, 1911—1917, INC. | |
|---|---|---|---|---|
| | | | Pneumonia | Pneumonia, Tuberculosis, Heart Disease and Nephritis |
| Columbus | 14.9 | 598 | 130.0 | 483.8 |
| Los Angeles | 12.5 | 559 | 90.8 | 588.9 |
| Indianapolis | 15.6 | 246 | 133.9 | 616.9 |
| Milwaukee | 12.2 | 537 | 133.2 | 406.0 |
| Rochester | 14.5 | 534 | 135.3 | 570.0 |
| Atlanta | 15.7 | 533 | 167.0 | 604.0 |
| Chicago | 14.4 | 524 | 188.0 | 610.8 |
| St. Paul | 11.0 | 510 | 132.6 | 411.0 |
| Toledo | 16.6 | 451 | 123.3 | 563.1 |
| Minneapolis | 11.9 | 437 | 109.8 | 451.4 |

Table X shows that ten of the twelve cities with the highest rates for pneumonia and heart disease had also excessively high rates for influenza and pneumonia during the three epidemic periods from January, 1917, to March, 1920. The exceptions were Fall River and Richmond and their rates were not far below the average.

TABLE X.

| CITY | PNEUMONIA AND HEART DISEASE | TOTAL EXCESS DEATH RATE, 3 EPIDEMIC PERIODS—JAN., 1917, TO JUNE, 1920 |
|---|---|---|
| Boston | 412 | 822 |
| Worcester | 412 | 754 |
| Pittsburgh | 403 | 1305 |
| Baltimore | 393 | 810 |
| New Haven | 384 | 765 |
| New Orleans | 381 | 854 |
| Nashville | 380 | 933 |
| Washington | 376 | 885 |
| Fall River | 375 | 673 |
| Richmond | 374 | 608 |
| Philadelphia | 367 | 1087 |
| San Francisco | 362 | 874 |

IMMUNITY.

Does an attack of influenza confer immunity and if so what is its duration?

This question is of the greatest interest and importance to the health officer but unfortunately, as in other phases of the subject of influenza, there is much conflicting testimony.

There has been a tendency to ascribe the relatively low mortality in the age groups over 40 to an immunity conferred by the epidemic beginning 1889-90 and lasting about three decades. Such an immunity would be expected to protect those above the age of 30. As a matter of fact the significant fall in the death rate is not apparent until after 40—the rate between 30 and 40 being almost as high as that from 20 to 30.

If such an immunity was actually conferred from 1889 to 1892 it is inconceivable that it would operate to protect those from ten to twenty years of age at that time and afford no protection to the group from one to ten years of age.

Arnold[17] made a study of persons attacked in Leicester, England, in the three epidemic waves—summer, 1918; autumn, 1918, and early in 1919, the peak week in these waves being the weeks ending July 20, November 2, and March 1. He concluded that considerable immunity to the autumn wave was exhibited by those who had suffered in the midsummer wave but that only slight immunity was evident in the February, March wave.

MacEwen[18] found that the percentage of those attacked in autumn in schools, colleges and among London police was much less among those who had had influenza in the summer wave. For example, of 1224 pupils in Finchley Council schools only 13% of those alleging an attack in the summer had influenza in the autumn, while 35% of those with no record of previous influenza were attacked.

Scoccia[19] states that all of the eighty nurses in the Spezia Hospital were attacked during May and June—late in September the epidemic recurred and not one of these 80 nurses was attacked.

Hamilton and Leonard[20] found that in a girls' school by strict quarantine the first outbreak in November, 1918, was confined to three cottage units. In January the disease returned but it spared those attacked in November.

Vaughan,[21] judging from experience in army

camps, states that the mild influenza of April, 1918, gave a marked degree of immunity against the epidemic of September, 1918.

Dr. Henry F. Vaughan, Commissioner of Health, Detroit, Michigan, estimates that in the period September, 1918, through March, 1919, that 125,000 cases occurred in Detroit or that 14% of the population was attacked.

If the 1920 epidemic was unaffected by immunity conferred in 1918-19, then 14% of the cases would probably occur among those previously attacked. Actually only 10% of the 1920 cases gave a history of a previous attack. From this Vaughan concludes that there was an effect in 1920 from the immunizing influence of the epidemic of 1918-19.

Frost found from a canvass in Baltimore of 4078 persons known to have escaped influenza in 1918 and 1059 known to have had influenza in 1918 that there was little difference in the percentage attacked in the two groups—and concludes from this that the 1918 attack did not confer immunity against attack in 1920 and that the immunity following an attack of influenza must be of short duration.

Judging from the results of his surveys in Baltimore, Frost believes that there is a transient immunity of a few months duration at least.

In analyzing the Baltimore epidemic he made a first canvass of 33,776 people between November 20 and December 11, 1918, and a second canvass of the same population in January to determine the extent of the recrudescence reported in December. Among 32,600 people included in this canvass, 724 cases of influenza were found to have occurred since the previous survey. Of this number, 121 cases were reported as second attacks, but on investigation through the attending physicians or by a medical officer, the clinical diagnosis of both attacks as influenza was confirmed in only 26 cases, or 0.37 per cent. of the total, and even in these cases the diagnosis is necessarily uncertain. Considering that 23 per cent. of the population had had influenza prior to December 11, the proportion of second attacks should have been much greater if no immunity had been acquired.

If the prepandemic rises shown on Chart II were due to the same disease as that responsible for the great pandemic rise of September, 1918, then we would expect that the rise in September, 1918, would be modified according to the number of people attacked in April, 1918—and the degree of immunity conferred by such attack.

What effect, if any, did the previous epidemics and especially the epidemic of March-April, 1918, have upon the prevalence of influenza in September-October, 1918? Table XI shows annual excess death rates, influenza and pneumonia, for certain cities for the following periods:

January, 1917, to September, 1918.
September, 1918, to June, 1919.
January to March 1920.

A careful study of this table shows certain striking features but no regularity in the relation between the four periods covered in the matter of prevalence.

1. Certain cities were hard hit in all periods, notably Pittsburgh, Philadelphia, Kansas City and Nashville.

2. Certain cities escaped with low rates in all periods—Minneapolis, St. Paul and Chicago.

3. Certain cities with high rates in the great pandemic had low rates in the other epidemic periods—Boston, Worcester, New Haven, Fall River and New Orleans.

4. Certain cities with low rates in the great pandemic and high rates in one of the other periods—Birmingham, Milwaukee, Columbus, St. Louis and Indianapolis.

The table does not show that the prepandemic waves had any definite or consistent influence on the prevalence in the great pandemic.

We must bear in mind, however, that mortality statistics alone do not accurately indicate prevalence unless we have knowledge of the ratio between cases and deaths also. Thus the prevalence in a mild epidemic might appear low because of a low case fatality rate, yet the reverse might be an actual fact. For this reason it is difficult to estimate the immunizing influence of an epidemic by mortality rates alone. A very mild epidemic with very few deaths might be very potent in immunizing large numbers of the population provided that an attack of the disease produced immunity. We cannot conclude, therefore, that a low mortality rate in April, 1918, necessarily means a low prevalence.

On the other hand, it is natural to conclude that the high mortality rates shown by Birmingham, Pittsburgh, Philadelphia, Nashville and Kansas City had apparently no effect on the pandemic prevalence although Birming-

ham had a relatively low prevalence in the pandemic of September-October, 1918.

These indications suggest one of two things: either that in the great pandemic we were dealing with a new and entirely different disease, or that the immunity conferred by an attack, if any, was of a very fleeting character,—a matter of less than six months.

TABLE XI.

ACTUAL EXCESS DEATH RATES PER 100,000 INFLUENZA AND PNEUMONIA (ALL FORMS) FOR EACH EPIDEMIC PERIOD.

| CITY | JAN., 1917 TO SEPT., 1918 | SEPT., 1918 TO JUNE, 1919 | JANUARY TO MARCH, 1920 |
|---|---|---|---|
| Albany, N. Y. | 33 | 631 | 64 |
| Atlanta, Ga. | 80 | 330 | 123 |
| Baltimore, Md. | 87 | 629 | 94 |
| Birmingham, Ala. | 238 | 497 | 120 |
| Boston, Mass. | 53 | 685 | 84 |
| Buffalo, N. Y. | 67 | 567 | 88 |
| Cambridge, Mass. | 62 | 532 | 65 |
| Chicago, Ill. | 25 | 404 | 95 |
| Cincinnati, Ohio | 52 | 531 | 67 |
| Cleveland, Ohio | 107 | 546 | 87 |
| Columbus, Ohio | 65 | 409 | 124 |
| Dayton, Ohio | 58 | 532 | 100 |
| Fall River, Mass. | 40 | 602 | 31 |
| Grand Rapids, Mich. | 38 | — | — |
| Indianapolis, Ind. | 67 | 354 | 125 |
| Jersey City, N. J. | 70 | — | — |
| Kansas City, Mo. | 132 | 689 | 222 |
| Los Angeles, Cal. | 7 | 505 | 47 |
| Louisville, Ky. | 113 | 530 | 58 |
| Lowell, Mass. | 97 | 548 | 91 |
| Memphis, Tenn. | 75 | 470 | 139 |
| Milwaukee, Wis. | 98 | 337 | 102 |
| Minneapolis, Minn. | 47 | 299 | 91 |
| Nashville, Tenn. | 135 | 662 | 136 |
| Newark, N. J. | 72 | 538 | 92 |
| New Haven, Conn. | 45 | 629 | 91 |
| New Orleans, La. | 35 | 761 | 57 |
| New York, N. Y. | 60 | 544 | 104 |
| Oakland, Cal. | 30 | 528 | 108 |
| Omaha, Neb. | 72 | 562 | 109 |
| Philadelphia, Pa. | 175 | 814 | 97 |
| Pittsburgh, Pa. | 238 | 891 | 176 |
| Providence, R. I. | 58 | 534 | 88 |
| Richmond, Va. | 40 | 520 | 48 |
| Rochester, N. Y. | 53 | 433 | 48 |
| St. Louis, Mo. | 103 | 406 | 120 |
| St. Paul, Minn. | 37 | 386 | 87 |
| San Francisco, Cal. | 33 | 724 | 117 |
| Syracuse, N. Y. | 60 | 580 | 147 |
| Toledo, Ohio. | 65 | 329 | 57 |
| Washington, D. C. | 80 | 689 | 116 |
| Worcester, Mass. | 42 | 618 | 94 |
| Average | 74.86 | 544.37 | 97.72 |

If an attack of influenza conferred immunity for a period of years we would expect an epidemic in April to modify a later epidemic in September of the same year.

Thus cities which are severely stricken in March-April, 1918, might be expected to escape with relatively low mortality in September-October, 1918.

On Chart X are plotted the excess death rates, influenza and pneumonia, all forms, in the epidemic periods, January, 1917, to September, 1918, and September, 1918, to June, 1919, for the forty large cities. The excess death rates were plotted as "relative figures" or variations from the mean, which is placed as 100 on the chart. The cities are arranged in the order of the severity of their death rate during the first of the two epidemic periods in order to bring out any correlation between the rates in the two epidemics.

The five cities which suffered most severely in the period January, 1917, to September, 1918, were Birmingham, Pittsburgh, Philadelphia, Nashville and Kansas City.

In the epidemic period September, 1918, to June, 1919, Birmingham escaped with a rate below the average—but Pittsburgh and Philadelphia were again hit hardest and Nashville and Kansas City were near the top of the list.

Louisville and Cleveland, too, were above the average for both epidemic periods. St. Louis and Milwaukee were more orthodox, being above the average for the first epidemic period and were well below the average in the second.

Baltimore, Washington and Atlanta were near the average for the first period. In the second period Baltimore suffered severely, Washington more severely, while Atlanta escaped with a low rate.

Many other examples of the contradictory results might be cited. Syracuse and Rochester both had low rates in the first period and Syracuse had an apparently compensatory high rate in the second, while Rochester again escaped with a low rate.

The best examples of cities which appear to give a compensatory high rate in the second period after a low rate in the first are New Orleans, San Francisco, and Boston. Striking examples of low rates in both epidemic periods are furnished by Toledo, Minneapolis, St. Paul, and Chicago.

Chart XI compares in the same manner the two epidemic periods—first, September, 1918, to June, 1919; second, January to March, 1920.

Here again while some cities seem to show the rates for the second epidemic to have been modified by the first, others suggest no such relation. New Orleans, Boston, New Haven, and Fall River, all with high rates in the period beginning September, 1918, have relatively low rates in 1920.

Birmingham, Memphis, St. Louis, Columbus, Indianapolis and Atlanta—with low rates in

the first period, had very high rates in the second.

On the other hand, Pittsburgh, Kansas City, San Francisco, Syracuse and Washington were severely stricken in both periods, while Oakland, Los Angeles, Rochester, St. Paul, and Minneapolis had low rates in both periods.

A careful study of Table XI shows many instances in which low and high rates alternate in successive epidemic periods, suggesting that immunity conferred in one epidemic period plays a part in the low rate which follows. Unfortunately for this conclusion quite a number of cities had high rates for all three periods so that charts X and XI do not show any consistent relation between a low rate in one epidemic period and a high rate in another.

It is possible that other reasons independent of immunity or lack of immunity may be found to explain why Pittsburgh, Nashville, Kansas City and Philadelphia had high rates in all periods.

## ETIOLOGY.

Two years after the epidemic of '89-90, Pfeiffer,[22] working with purulent bronchitis and broncho-pneumonia, observed and cultivated small gram negative bacilli, which he previously had seen in the sputum of cases during the great epidemic. He demonstrated that these organisms could not be cultivated by the methods used by other workers in the epidemic of 1889 and this fact was held to explain failure to isolate during 1889. Pfeiffer felt justified in attributing the 1889 epidemic to this cause which he named the influenza bacillus.

Pfeiffer claimed that he found influenza bacilli in all fresh uncomplicated cases of influenza. He also claimed that they were found only in cases of influenza, acute or convalescent. He described what he called pseudo-influenza bacilli—larger than influenza bacillus and with a tendency to thread formation, as occurring in broncho-pneumonia complicating diphtheria.

Weichselbaum[23] confirmed Pfeiffer's findings in 1892, and in 1897 Grassberger[24] published similar findings. The French[25] workers did not accept the claims of Pfeiffer.

Many workers have since been unable to demonstrate the existence of pseudo-influenza bacilli as a group. Since Pfeiffer's time and until the recent pandemic the great majority of workers have failed to find the influenza bacilli in cases of influenza in such high percentages as reported by Pfeiffer and have found B. influenzae in those suffering from other diseases and in healthy persons.

## IS THE B. INFLUENZAE THE CAUSE OF INFLUENZA?

The experience of workers in the great pandemic and since September, 1918, has resulted in very conflicting reports concerning the etiology of influenza. While Pfeiffer's claims have received some new support, many believe B. influenzae to be only a secondary invader.

Abrahams[26] considers the primary infection to be due to B. influenzae and that pneumococci, streptococci, or diplostreptococci are secondary invaders, producing the fatal result. Rucker and Werner[27] held a similar view. Keegan[28] recovered B. influenzae from 86% of necropsies and considers it the primary cause.

Spooner, Sellards and Wyman,[29] working at Camp Devens, consider that B. influenzae is established as the cause of the epidemic. The Kitasato Institute[30] findings are that the influenza bacillus of Pfeiffer was the cause of the 1918 pandemic.

These writers practically all believe that the initial damage is done by the influenza bacillus which paves the way for secondary invaders.

Lucke, Wight and Kime[31] report frequent finding of B. influenzae and believe that if not the primary cause it is an indication of influenza.

Duval and Harris[32] believe B. influenzae is the cause of influenza and claim to have found specific immune bodies to B. influenzae in the blood of influenza patients during and after infection.

Small and Stangl[33] in the 1920 epidemic in Chicago found B. influenzae in 100% of acute influenza patients. In the pneumonia cases studied they found pneumococci in 84%; hemolytic streptococci in 18.7% and B. influenzae in 75%.

Many observers finding B. influenzae in acute influenza in a very high percentage of cases believe that faulty technique may explain failure to find in all cases. Park evidently holds such a view and Keegan suggests the same explanation of some of the failures to find in high percentages in the early acute stage.

Wade and Manalang[34] report an interesting variation in morphology of the Pfeiffer bacillus:

"It has been found that three different strains of an organism supposed to be Bacillus influenzae will, under certain conditions, aban-

don the usual bacillary form and grow as a frank fungus, morphologically of the Dicomyces type. Under other conditions they show less modification, the most striking feature then being the production of condiospores, bodies of a type not found in true bacteria. That this organism may not be the true Pfeiffer bacillus is conceivable, of course, but considering the source, morphology, ordinary cultural characteristics, and the poison production of the one strain tested, we consider this highly improbable. Further, we are confident that the cultures do not contain any contaminating organisms, as may be suggested. In short, we believe that we have been dealing solely with the true Pfeiffer bacillus."

It is interesting to note in connection with the results of Wade and Manalang cited above that Pfeiffer described what he called pseudo influenza bacilli with a tendency to thread formation.

Jordan[35] found the Pfeiffer bacillus in 64% of cases examined October, 1918, to February, 1919. The other organism most commonly found was the Mathers streptococcus. He found the pneumococcus in 20%. He states:

"The observation carried out by the aerobic blood-agar plate method and recorded in this paper have not shown the predominance or constant presence of any one organism in the upper respiratory tract of influenza patients. The Pfeiffer bacillus, however, has been more conspicuous than any other organism, particularly in comparison with its relative infrequency in cases of rhinitis and tonsillitis examined during the same epidemic period."

Albert and Kelman[36] find that "the influenza bacillus produces a toxin which is fatal to mice, guinea-pigs and rabbits almost as rapidly as are broth cultures of equal dosage. This toxin is produced very rapidly and can be obtained by filtering broth cultures. It is not possible to state definitely whether it is an endotoxin or an extracellular one.

"Although the symptoms of intoxication as seen in lower animals following injections of the Pfeiffer bacillus are suggestive of the profound intoxication seen in connection with many cases of the epidemic disease influenza in the human being, these experiments do not furnish any proof that the Pfeiffer bacillus has any specific etiologic relationship to that disease. On the other hand, they suggest that a possible etiologic relationship cannot be ignored."

Huntoon and Hannum[37] claim that B. influenzae produces a toxin which produces in animals congestion of the respiratory tract with hemorrhages into the alveoli.

Ferry and Houghton[38] report similar findings in regard to a toxin and claim to have produced an antitoxin which protected guinea pigs.

Roos[39] also reports on the toxin production of B. influenzae and reports similar results to Huntoon and Hannum in the effect of the toxin in predisposing to invasion by secondary organisms.

Another very large group believes that some unknown organism causes the initial damage by breaking down the natural protective barriers and permitting secondary invasion by the pneumococci, streptococci and B. influenzae, which they consider as a secondary invader only.

Park[40] says:

"These results appear to us to throw the influenza bacilli in the cases studied as clearly into the class of secondary invaders.

"We believe that the other microörganisms, such as certain streptococci and pneumococci which are under suspicion in different localities will be found after subjection to similar severe tests not to possess the necessary identity of characteristics to allow them to remain under serious consideration as the primary agent in this epidemic, but rather like the influenza bacillus, to be reckoned among the most important of the secondary invaders.

"Our final conclusion is, therefore, that the microörganism causing this epidemic has not yet been identified."

Howard[41] also believes that the influenza bacillus is a secondary invader only, which may produce a terminal broncho-pneumonia.

Wollstein,[42] working with sera of convalescents from influenza concludes:

"The patients' serological reactions indicate the parasitic nature of the bacillus, but are not sufficiently stable and clean-cut to signify that Pfeiffer's bacillus is the specific inciting agent of epidemic influenza. They do, however, indicate that the bacillus of Pfeiffer is at least a very common secondary invader of influenza, and that its presence influences the course of the pathological process."

Sellards and Sturm[43] report finding an or-

ganism with all the characteristics of B. influenzae in a series of measles cases in both sputum and conjunctivae with the disappearance of the organism with subsidence of symptoms in three-fourths of the cases. They consider that presence of B. influenzae in two diseases shows a causal relation to neither.

THE RÔLE OF STREPTOCOCCI, PNEUMOCOCCI AND OTHER ORGANISMS.

There seems to be relative unanimity of opinion as to the rôle played by the pneumococci, streptococci and other organisms commonly found in the upper air passages. Practically all observers consider these as secondary invaders.

Many writers[44] have associated streptococci with the complications of measles and have stressed the likelihood of streptococcus carriers suffering severely from those complications. This is suggestive of what may happen in influenza with both streptococci and pneumococci.

Hall, Stone and Simpson[45] found pneumococci in influenza sputa in 302 cases, 273 of which were Type IV.

Blanton and Irons[46] found streptococci in 451 and Type IV pneumococcus in 148 cases out of a total of 749. Thirty per cent. of the streptococci were hemolytic, 70% non-hemolytic. Opie[47] and his co-workers found pneumococci in 61.2% of those examined. They believe that the fatal factor was a lobar pneumonia, and that the streptococcus hemolyticus played an insignificant part in the production of pneumonia.

In their second report these authors modify their statement in regard to the significance of streptococci:

"The sequence of events that occurs in many cases of influenza is as follows: B. influenzae descends into the bronchi; pneumococci (in this camp usually Type IV) invade the inflamed bronchi; enter the lung, and produce either lobar pneumonia or bronchopneumonia. Hemolytic streptococci may descend and infect the pneumonic lung. It is not improbable that hemolytic streptococci may invade the bronchi previously infected with B. influenzae and cause bronchopneumonia in the absence of pneumococci.

"When hemolytic streptococci invade the lung either with or without preceding pneumococcus infection, there may be no suppuration of the lung. It is probable that death has occurred before there is opportunity for the formation of abscesses. Streptococcus hemolyticus may pass through a pneumonic lung and appear in the heart's blood although there has been no suppuration. It is not improbable that it may pass through the lung and produce empyema, the lung remaining free from actual suppuration. Lobar pneumonia appears to resist suppuration more effectively than bronchopneumonia; but fatal streptococcus infection is common with both.

"Infection with hemolytic streptococci may spread as an epidemic through the pneumonia wards of a hospital. A single patient with streptococcus pneumonia is a source of grave danger to every patient in the same ward. Superimposed infection with hemolytic streptococci increases the mortality of pneumonia so that it may reach from 50 to 100 per cent. of all patients with pneumonia."

Birge and Havens[48] found streptococcus hemolyticus in 60 cases ante mortem and in many post mortem cases of influenza pneumonia.

Goodpasture[49] in the later stage of the epidemic found streptococcus hemolyticus in all of the 16 cases which came to necropsy.

Tunnicliff[50] states that Mathers found a green producing streptococcus in 87% of 110 cases. The same writer declares that specific opsonins develop in the course of influenza, but that with onset of pneumonia these opsonins decrease. The changes in opsonic power are specific for the green producing coccus and no fluctuations being observed for streptococcus hemolyticus, B. influenzae or M. catarrhalis.

Rosenow[51] from immunologic studies believes that there is a pandemic strain of green producing diplostreptococci in influenza. He produced results in guinea pigs similar to the pathologic changes in human lungs in influenza. Rosenow also claims to have found evidence in group reaction between green producing and hemolytic strains of streptococci.

Dochez, Avery and Lancefield[52] claim that immunological differences have been shown to exist between strains of streptococcus hemolyticus of the human type and that four biological types have been identified by means of the reactions of agglutination and protection and that at least two other types have been encountered and the indications are that more exist.

Jordan[53] found the Mathers' coccus about

as frequently as B. influenzae and states that its association with pneumonia cases seemed to be closer than that of the Pfeiffer bacillus.

Howell and Anderson[54] in a complement fixation experiment conclude:

"The outstanding feature of this work on complement fixation with influenza serum is the large number of positive results with certain strains of the viridans group of streptococci isolated from cases of influenza at Camp Meade and in Chicago. The evidence indicates that such organisms probably played an important part in the morbid process even in other places. Serum from influenza patients in several different places appears to have acquired similar new properties."

Claims that a filterable virus is the primary cause of influenza have been made by many writers. Nicolle and Lebailly,[55] de la Rivière,[56] da Cunha, Magalhaes, and da Fonseca,[57] and Gibson, Bowman, and Connor[58] have reported transmission of the disease by filtrates, and von Angerer,[59] da Cunha, Magalhaes, and da Fonseca, Leschke,[60] Bradford, Bashford, and Wilson,[61] and Gibson, Bowman, and Connor,[62] claim to have cultivated minute filterable organisms.

Wilson[63] made the following claims:

1. An organism, of definite morphological and cultural characters, has been isolated from cases of influenza.

2. It can be demonstrated in the blood, sputum, and other exudates, and in the tissues, post mortem, by appropriate methods of staining.

3. It belongs to the group of "filter-passers," a group of organisms which pass through bacteriological filters. It has been seen microscopically in the filtrate and has been cultivated therefrom.

4. It has not been found in a large series of controls.

Arkwright[64] took exception to these claims after an attempt to confirm them and as a result Wilson modified the claims by withdrawing the claim that a filter passing organism had been grown in pure culture.

Other workers, notably Rosenau,[65] have failed to transmit the disease by filtrates. Rosenau had 100 young volunteers of the most susceptible age. He used suspensions of Pfeiffer's bacillus, citrated blood of influenza patients, emulsion of sputa unfiltered and filtered mucous secretions. All results were negative.

McCoy had similar results at San Francisco with enlisted men of the Yerba Buena Naval Training Station. These experiments do not definitely rule out a filterable virus and the results are particularly surprising in a disease which in the field of epidemic times appears to be very readily transmissible.

Wahl, White and Lyall[66] failed to transmit the disease to man using the filtrate from a pneumonic lung directly into the subject's nasal passages. They had negative results also with emulsions of B. influenzae.

## CONCLUSIONS.

It is probable that influenza is a disease of great antiquity and that the cause of the world-wide pandemics and interpandemic outbreaks is the same.

With a strong predilection for the winter months we have influenza with us every year—and in retrospect we can detect in the mortality statistics outbreaks reaching epidemic proportions in 22 out of the 30 years since 1889.

In 1918-19 the attack rate varied from 15 to 40% and seemed to be highest in the age group five to nine—declining in each successive age group except 25 to 34, which exceeded the rate for the group 15 to 24.

The incidence in 1918-19 was greater in females than males and the disparity was most noticeable in the ages from 25 to 40, indicating, according to Frost, that the females from 15 to 45 were either more susceptible or more intimately exposed to infection than males of corresponding age.

Case fatality in the 1918-19 epidemic was about 2% and was slightly higher in females under 15 and very much higher in females over 60 than in males of corresponding ages. From 15 to 60 the case fatality was much higher in males.

There was great variation in 40 large cities in explosiveness of the epidemic and in the severity as measured by the excess death rates for the entire epidemic period.

There seemed to be some correlation between explosiveness and the severity as measured by excess death rates—the greatest mortality being usually but not always in cities with a high explosive index.

There was little consistency in the explosiveness of the two epidemics, 1918-19 and 1920 upon comparing the indices in the various cities. Cities with a high explosive index in

1918-19 often had a low index in 1920. Most cities with a high explosive index for 1920 had a low index for 1918-19. Memphis, Nashville, and Washington had a high index of explosiveness in both epidemics.

There seemed to be some correlation between explosiveness and the general death rate and the rates for the four principal causes of death —pneumonia, tuberculosis, heart disease and nephritis. There seemed to be considerable correlation between the total excess death rates for the epidemic periods and the general death rate and the death rates for pneumonia, tuberculosis, heart disease and nephritis.

All the evidence points to an immunity of relatively short duration—probably of months rather than years.

The etiologic cause is unknown. There is not sufficient evidence to warrant the view that B. influenzae is anything more than a secondary invader. The claims for a filterable virus are strong but much additional work will be necessary to make certain many things which are now only possibilities.

A survey of the whole field and all available literature convinces me that while further epidemiologic studies will have great value and be of intense interest they will not furnish a solution of the problem. We must have more intensive, comprehensive and sustained laboratory research, using the body fluids and secretions of influenza cases for material if we hope to solve the problem and secure the biologic aids which we now lack for the prophylaxis and treatment of influenza.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## Clinical Department.

### PNEUMONIA IN A WOMAN WITH AN HABITUAL HIGH BLOOD PRESSURE.

BY M. J. KONIKOW, M.D., BOSTON.

DURING the last influenza epidemic I came across a pneumonia case that presents a peculiar interest to the profession, as it occurred in a female patient of 54, whose habitual systolic blood pressure was during the last two years never less than 225,and at times reached as high as 275.

The following table records the temperature, pulse, respiration, and blood pressure from February 24, 1920, her first day of sickness, to March 21, 1920, my last visit, when she was discharged as recovered from the attack of pneumonia.

| Date | Temperature A.M. | Temperature P.M. | Pulse A.M. | Pulse P.M. | Respiration A.M. | Respiration P.M. | Blood Pressure D. | Blood Pressure S. |
|---|---|---|---|---|---|---|---|---|
| Feb. 24, 1920 | 100 | | | | | | 100 | 100 |
| 25, | 101 | | | | | | 90 | 155 |
| 26, | 104 | | | | | | {80 | 140* |
| 27, | 104 | | | | | | {80 | 130† |
| 28, | 103.8 | 104.2 | 100 | 108 | 35 | 40 | 90 | 150 |
| 29, | 103.0 | 104.0 | 96 | 104 | 35 | 37 | 95 | 145 |
| March 1, | 103.3 | 102.9 | 96 | 92 | 37 | 28 | 90 | 170 |
| 2, | 102.0 | 100.5 | 94 | 98 | 35 | 32 | 90 | 180 |
| 3, | 99.6 | 98.8 | 90 | 90 | 30 | 32 | 90 | 180 |
| 4, | 98.8 | 100.5 | 90 | 90 | 32 | 32 | 90 | 180 |
| 5, | 99.0 | 100.0 | 94 | 90 | 30 | 30 | 90 | 190 |
| 6, | 98.2 | 99.5 | 90 | 90 | 30 | 28 | 90 | 190 |
| 7, | 98.4 | 99.2 | 90 | 94 | 28 | 28 | 90 | 195 |
| 8, | 98.0 | 99.0 | 94 | 94 | 25 | 25 | 90 | 205 |
| 9, | 97.8 | 98.5 | 96 | 96 | 24 | 24 | 90 | 205 |
| 10, | 98.1 | 98.3 | 92 | 90 | 23 | 26 | 90 | 195 |
| 11, | 97.6 | 98.6 | 94 | 94 | 22 | 20 | 105 | 200 |
| 14, | 98.2 | 98.6 | 92 | 88 | 20 | 20 | 105 | 200 |
| 17, | 98.0 | 98.2 | 90 | 94 | 20 | 20 | 110 | 220 |
| 21, | | | | | | | | |

* A.M. † P.M.

As it is seen from this table, the first blood pressure was taken on February 29, probably on the third day of the pneumonia. To my astonishment, this blood pressure fell from her habitual 250 (S) to 160 (S), and continued to fall until it reached, on March 2, as low as 80/130, when the lysis began and the dullness reached its height. From this date with the gradual fall of the temperature, of the pulse, and of the respiration, the blood pressure began to climb again, the diastolic slowly, the systolic quite rapidly, until it reached, on the twenty-first of March, 100/230, almost to her habitual blood pressure.

The tragi-comical side of this case is the fact that while during the past two years attempts were made by many physicians, myself included, to reduce this extremely high blood pressure, and always with a negative result, influenza-pneumonia, with its toxins, began to lower the blood pressure, until it brought it down to 130. What could not be done in two years, the influenza pneumonia achieved in three days. Do we rejoice at this low blood pressure? Certainly not. We tremble for the patient's life, as this low blood pressure simply signifies the weakening of the heart's muscle, the inability of the heart to take proper care of the blood circulation. And as this blood pressure begins to rise, we welcome the rise as the surest sign of approaching recovery, and when at last it reaches the habitual pre-pneumonia height, we know the patient has recovered.

Moral: Leave the high blood pressure alone, if the cause of it cannot be ascertained and removed. High blood pressure in itself is merely nature's means of self-defence.

## Book Reviews.

*Vital Statistics.* By GEORGE CHANDLER WHIPPLE. New York: John Wiley and Sons, Inc. 1919.

The science of demography, the application of statistical study to human life, will undoubtedly develop and become increasingly important in the future as it becomes necessary to condense and make usable the knowledge which will be gathered by public health officials. The publication of this small volume dealing with "Vital Statistics" comes at an opportune time, when nations have become aware, through the Great War, of the necessity of knowing more accurately the conditions in their own and in other countries. The science of demography includes the study of genealogy, human eugenics, the collection by census of social, political, religious, and educational facts concerning population, registration of vital facts, the application of the statistical method to the study of these facts, biometrics, and statistical pathology. The object of this book is to explain how to collect and interpret data on these subjects. It considers statistical arithmetic methods, and how to express vital facts by figures, how to tabulate them, and how to present them in diagrammatic form. Methods of estimating population, of computing birth-rates, marriage-rates, death-rates, and life-tables, of classifying diseases and analyzing death-rates are explained. A chapter dealing with the problem of correlating relations between series, classes, or groups of data, with a view to determining cause and effect, is particularly significant in public health work. In order to make the subject matter more convenient for school instruction, exercises and questions are given at the end of every chapter to incite further study. In the training of future health officers to use vital statistics with unquestionable ability, this book will be found extremely valuable.

*Skin and Venereal Diseases.* The Practical Medicine Series. Volume VII. Edited by OLIVER S. ORMSBY, M.D., and JAMES HERBERT MITCHELL, M.D. Chicago: The Year Book Publishers. 1918.

The purpose of the Practical Medicine Series is to review in a series of eight volumes, issued at monthly intervals, the entire field of medicine and surgery for the year preceding its publication. Although this series is published primarily for the general practitioner, it is so arranged that persons interested in special subjects can obtain in single volumes information on those subjects. Volume VII deals with *Skin and Venereal Diseases.* It contains a statistical report of the American Dermatological Association for the years 1912-1916 and reports of recent research work, such as investigation of the nature and formation of pigment, skin ferments, the causes and treatment of eczema, and treatment of dermatitis venenata by vegetable toxins. Diseases of the scalp and nails, tropical dermatoses, dermatoses due to external irritants, and infectious dermatoses are considered from the point of view of both diagnosis and treatment. In the part of the book devoted to venereal diseases, methods of controlling venereal disease in the army are explained, and the characteristics of gonorrhea, syphilis, chancroid, bubo, and other diseases of venereal origin are considered. The teaching of syphilis is an important problem at the present time, and this volume contains valuable suggestions for organizing syphilis departments and equipping clinics. This volume of the series will be as helpful to the profession as its predecessors.

*Nervous and Mental Diseases.* The Practical Medicine Series, Volume VIII. Chicago: The Year Book. 1919.

The Practical Medicine Series aims to cover the whole field of medicine and surgery for the year preceding its publication. It is so arranged that, although designed primarily for the general practitioner, persons interested in special subjects may obtain in separate volumes the works which to them are of particular interest.

Volume VIII reviews the subject of *Nervous and Mental Diseases.* The war has to a great extent determined the general course of work carried on in neurology and psychiatry during the past year, and from this source, abundant material of unusual and permanent value has been obtained; problems arising from neuroses and psychoses of war and wounds of all parts of the nervous system are considered. This book describes, also, diseases which have been prevalent during the year in epidemic form, one of the most interesting of which has been the "lethargie" encephalitis occurring in Europe and Australia. Among the monographs not connected with the war or epidemic conditions, perhaps the most important is Cushing's article on "Tumors of the Acoustic Nerve."

This volume, like the preceding publications of this series, presents in concise form the most recent information relating to the subjects with which it is concerned.

*United States Army X-ray Manual.* New York: Paul B. Hoeber. 1918.

The need for a large number of roentgenologists for service in military hospitals has made it necessary to train for this work many physicians who have had little or no practice in this field. To facilitate the rapid training of these men, this textbook, *United States Army X-ray Manual,* has been prepared under the direction of the Surgeon General. Although this book is not a complete treatise, it covers practically all phases of roentgenology, with particular emphasis on x-ray diagnosis. The purpose of this book is to familiarize the roentgenologist with the apparatus with which he must work under war conditions, and to give him further training in those parts of the work in which he is deficient. As the roentgenologist in the forward hospital is concerned for the most part with localization of foreign bodies, the part of the book devoted to localization is of particular value. The new apparatus designed for use in war service is described in detail, with consideration of its limitations and advantages. The two hundred and nineteen illustrations which are included in this volume are unusually instructive.

*The Eye, Ear, Nose, and Throat.* The Practical Medicine Series, Volume III. Edited by CASEY A. WOOD, C.M., M.D., D.C.S.; ALBERT H. ANDREWS, M.D.; GEORGE E. SHAMBOUGH, M.D. Chicago: The Year Book Publishers. 1918.

This book is the third of a series of eight volumes of the The Practical Medicine Series, issued for the purpose of summarizing the year's progress in medicine and surgery. This volume is divided into three parts, dealing with the eye, ear, nose, and throat, and contains extracts from the most important contributions to literature on these subjects. Chapters dealing with military surgery of the eye and military otology are of particular value at the present time. Although the book is comprised of information gathered from many sources, it is written concisely and in compact form, and offers to the general practitioner and student a complete review of the subjects with which it is concerned.

THE BOSTON
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BOSTON MEDICAL AND SURGICAL JOURNAL
126 Massachusetts Ave., Cor. Boylston St., Boston 17, Massachusetts

## RENEWAL OF NARCOTIC DRUG REGISTRATION.

FORMS for the renewal of registration under the Harrison narcotic law are being distributed from the Internal Revenue office to dealers, practitioners, and druggists, for the period of one year, commencing July 1. In order to comply with the provisions of the law these forms should be executed and returned to the Internal Revenue office on or before July 1 with payment of the tax.

In sending out these forms it is necessary that persons registering in one of the first four classes must also register in Class 5 in order to dispense or deal in exempt preparations and remedies, although the payment of an additional tax is not required. A separate return must be filed, however, for each class to which a person belongs.

Persons registered in either class 1 or 2 are not required to file an annual inventory as the monthly returns which they submit show the quantity of narcotic drugs on hand at the end of each month. Those registered in classes 3 and 4, and class 5 (manufacturers only), must prepare an annual inventory of all narcotic drugs and preparations on hand (excluding exempt preparations and remedies), one copy of which is to be submitted to the Internal Revenue office.

## USE OF ARSENIC PREPARATIONS IN THE TREATMENT OF SYPHILIS.

ON account of the extensive exploitation through advertisements in professional journals and otherwise of various arsenic preparations which are not related to the arsphenamine group, the United States Public Health Service has issued for the benefit of physicians a warning advising against the indiscriminate use of untried preparations in the treatment of syphilis. Attention is invited to the fact that provision has been made for the experimental use of any preparation under conditions which will make the results of such experiments available to others than the physician immediately concerned.

It is the opinion of the Bureau of Public Health Service that in the interest of all concerned, the subcutaneous, intramuscular or intravenous use of arsenic in the treatment of syphilis should be confined to preparations of the arsphenamine group, as these agents are of established value and are produced under the regulations of the Public Health Service. The following firms are now licensed for the manufacture of arsphenamine and neo-arsphenamine: Dermatological Research Laboratories, Philadelphia; H. A. Metz Laboratories, New York City; Diarsenol Co., Inc., Buffalo, New York; Takamine Laboratories, Clifton, New Jersey. The Lowy Laboratory, Newark, New Jersey, has been granted a license to prepare a stable solution of arsphenamine.

It is not the desire of the Bureau to limit clinicians in the choice of agents of recognized worth but in the case of arsenic preparations, not members of the arsphenamine group, the available evidence indicates that their routine use is inadvisable in the treatment of syphilis. If it is desired to use any of these preparations in a purely experimental way, previous authority from the Bureau should be secured. Appli-

cations for this authority should be accompanied by a statement as to the composition of the drug including the structural formula and the reason for its use. All information available on the value of the preparation should be forwarded.

## ANNUAL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY.

The one hundred and thirty-ninth annual meeting of the Massachusetts Medical Society was held in Boston on June 8 and 9, at the Boston Medical Library, with an attendance of five hundred medical men and women. Clinics were held during the morning of June 9, there were Medical Sections in the afternoon, and the Shattuck Lecture, "Influenza," delivered by Dr. Allan J. McLaughlin, was largely attended in the evening. Dr. McLaughlin's address will be published in a later issue of the JOURNAL.

On the morning of June 9 the regular annual meeting of the Society was held, preceded by a meeting of the Council and followed by the "Annual Discourse," by Dr. Hugh Cabot. Dr. Cabot's lecture, "Health Insurance, State Medicine, or What?" was published in the issue of the JOURNAL for June 10. The regular proceedings of the Sections and of the Society will be published, as usual, in subsequent issues of the JOURNAL. The afternoon was devoted to regular Section meetings. On the evening of June 9 the annual dinner was held at the American House, followed by a play, "It's a Great Life if You Don't Weaken," given by members of the Norfolk District Medical Society.

The officers of the preceding year were re-elected, with Dr. Frederick E. Jones as the new vice-president.

## MEDICAL NOTES.

OFFICERS OF THE ASSOCIATION OF AMERICAN PHYSICIANS.—At the annual meeting of the Association of American Physicians held in Atlantic City, the following officers were elected for the ensuing year: President, Dr. William S. Thayer, Baltimore; Vice-President, Dr. Herbert C. Moffitt, San Francisco; Secretary, Dr. Thomas McCrae, Philadelphia; Recorder, Dr. Thomas R. Boggs, Baltimore; and Treasurer, Dr. Joseph A. Capps, Chicago.

AWARD OF BRITISH HONOR TO MAJOR GENERAL WILLIAM C. GORGAS.—A knight commandership of the Order of St. Michael and St. George has been conferred by King George on Major General William C. Gorgas, former surgeon General of the United States Army.

DRUG PRICE CHANGES.—An announcement made on June 9 stated that prices in pharmaceutical drugs have reached their height and may be expected to become lower in a short time, although it may be many months before many important commodities go back to prewar averages. During the latter half of May and the first half of June there has been considerable cutting in retail prices, chiefly among second, third, or fourth-hand dealers. With a few exceptions such as alcohol, ether, glycerine, acetanilid, acetphenetiden, oxylates, caffeine, phenolphthalein, santonine, saccharine, and a number of the essential oil products, the general tendency of the market has been downward. It is believed by many that Teutonic competition will begin to figure more prominently during the summer months and that by fall a number of leading commodities will be affected by it.

SANATORIUM FOR TUBERCULOUS SOLDIERS.—The tuberculosis sanatorium heretofore operated by the army authorities at Fort Bayard, New Mexico, has just been transferred to the U. S. Public Health Service, and will soon be available for treating discharged, disabled soldiers. This sanatorium will provide the Public Health Service with 1,000 additional beds to care for its tuberculous patients. The present sanatorium at Deming will be held in reserve, especially for winter use.

At the Fort Bayard Sanatorium the Public Health Service will treat only ambulatory cases of tuberculosis, in which the prognosis is favorable. Patients will be admitted only after careful observation elsewhere to make sure that their condition is suitable for successful treatment at the high altitude of this sanatorium. In general, it is the policy of the Public Health Service not to move patients far from their homes, for experience has shown that such removal often has an unfavorable effect. For

this reason patients for the new sanatorium will probably be drawn principally from the middle and south-west sections of the country.

CANADIAN MEDICAL ASSOCIATION.—The fifty-first annual meeting of the Canadian Medical Association was held at Vancouver, British Columbia, on June 22, 23, 24 and 25, 1920. The presidential address was delivered by R. E. McKechnie, M.D., C.M., F.A.C.S., Chancellor of the University of British Columbia, Vancouver, B. C. Sections were held in Medicine, Surgery, Gynecology and Obstetrics, Ophthalmology and Otolaryngology, Orthopedics, X-ray, Genito-urinary Diseases, Child Welfare and Public Health. Among the addresses were included the following: In Medicine, "Certain Fundamental Errors in the Diagnosis and Treatment of Myocardial Insufficiency," by Charles Lyman Greene, M.D., St. Paul, Minnesota; in Surgery, "The Surgical Treatment of Ulcerated Intestinal Tuberculosis as Occurring Chiefly in the Course of Pulmonary Tuberculosis," by Edward W. Archibald, M.D., Montreal, Quebec; in Orthopedics, "Development and Scope of Orthopedic Surgery," by V. P. Gibney, M.D., Ottawa, Ontario; in Public Health, "The Federal Government and Public Health," by John Amyot, M.D., Ottawa, Ontario. The program of the Section of Medicine included a symposium on "Goitre" on June 23 and one on "Stomach and Duodenum," on June 25. The Surgical Section held a symposium on "Pulmonary Abscess" on June 23.

Also, there were meetings of the Canadian Public Health Association, The Canadian Association for the Prevention of Tuberculosis, The National Committee for Combating Venereal Diseases, and The Canadian Committee on Mental Hygiene. The British Columbia Hospital Association Convention was held in Vancouver at the same time as the meeting of the Canadian Medical Association.

SPREAD OF BUBONIC PLAGUE.—The bubonic plague has spread from Vera Cruz to Tampico, Mexico. Aid in fighting the disease has been asked of the Government of the United States and of the American Red Cross, and vaccine and other medical supplies have arrived already at Vera Cruz from this country. Of the twenty-four cases of bubonic plague reported, twenty-three have proved fatal. The Mexican federal authorities have stopped traffic with the interior by destroying railway tracks for five miles on all lines leading out of the city of Vera Cruz.

A report has been received from Pensacola, Florida, of a death from bubonic plague in that city, and public health officials have undertaken an investigation to check the spread of the disease there. Efforts will be made to exterminate rats, and special precautions will be taken so that no ships dock at Pensacola without proper rat guards.

### BOSTON AND MASSACHUSETTS.

MASSACHUSETTS SOCIETY FOR MENTAL HYGIENE.—The Massachusetts Society for Mental Hygiene was organized and incorporated in 1913 and began its public service in 1914 under the presidency of the late Judge Harvey H. Baker. During the five years of its existence the Society has endeavored to prevent mental disease and defect by educating the community in the principles of mental health, by fostering the mental health of normal children, by protecting the adolescent from mental and nervous breakdown, by the intelligent treatment of the feeble-minded, by improving the standards of care for those suffering from or in danger of developing mental disorder, promoting the study of mental disease and defect in their various forms and in their social and economic relations, and by disseminating knowledge concerning their causes, treatment, and prevention.

Since the organization of the Massachusetts Society for Mental Hygiene the number of persons who have learned to understand the problems of the mentally disordered and defective has been greatly increased, with a consequent increasing demand for adequate care and treatment for these members of society. The distribution of 177,546 publications during the last few years has assisted substantially in the dissemination of important information concerning mental health and diseases. Public conferences and exhibits have been held for the discussion and illustration of facts relating to mental health, individual efficiency, war neuroses, the extent, nature, causes, and means of prevention of mental disease, and the modern methods of care and treatment. During the war the Massachusetts Society, as a coöperative unit of The Medical Committee for Mental Hygiene, shared in examining recruited officers

and men for the detection of nervous and mental disorders.

One of the most important branches of service of the Massachusetts Society for Mental Hygiene is its lecture service. During the war this was interrupted; but up to that time two hundred and thirteen lectures had been given by physicians and social workers throughout the State under the auspices of the Society. This lecture service has been resumed and at present covers many phases of mental hygiene. Through the Society, speakers are provided without cost, except for necessary travelling expenses. Arrangements for lecturers should be made through the Medical Director of the Society, Dr. A. Warren Stearns, 1132 Kimball Building, 18 Tremont Street, Boston.

In a report published this year by the Massachusetts Society for Mental Hygiene have been outlined the following needs of mental hygiene in Massachusetts at the present time: to create and maintain an enlightened public opinion concerning the relations of mental normality and abnormality to a useful life in the community; to maintain the highest standard in our state institutions; to foster research and investigation tending to increase the knowledge of this subject and in that way ultimately to reduce burdens; to extend the investigations now being made in a few centers on the relation between mental disease, personality, and crime; to take an advanced stand concerning the prevention of feeble-mindedness; to extend special classes now so successful in a few places; to formulate and to carry out organized effort for the care of the handicapped in the community through social service; to promote the establishment of courses on mental hygiene in the professional and normal schools; to emphasize the need of mental hygiene in the industries.

Bequests to Medical Institutions.—The will of the late Mr. Daniel F. Chase of Quincy provided for the gift of two thousand dollars each to the Boston Floating Hospital, Sharon Sanatorium, and the Quincy City Hospital.

The Goddard Hospital.—The Goddard Hospital in Brockton was established in 1902 by the late Dr. Henry E. Goddard, primarily for the care and treatment of obstetrical cases, but later for both surgical and obstetrical cases. During the eighteen years of its existence the hospital has grown rapidly, expanding from its original headquarters with one room in a private dwelling to the present new hospital with a capacity of thirty-six beds. All the rooms are private or semi-private, there being no open wards. In July, 1919, the hospital was incorporated under the Massachusetts laws, and Dr. Samuel W. Goddard was elected president of the institution. The hospital is conducted on the Mayo plan with a limited closed staff, each member of which is following a special line of work.

The annual report for 1919 states that 752 cases were admitted to the Goddard Hospital during the year; this figure represents the largest increase made in any year. As it has been impossible for the hospital to care for all the patients who have desired to enter, during the last six months a waiting list has been established. When the new hospital building was opened it was intended to institute a medical service, but the heavy demands on the surgical and obstetrical departments have made this impossible and only an occasional case of this type has been admitted. The medical work is designed to secure the most efficient treatment for patients entering the surgical and obstetrical departments, having in view the following objects: (1) to safeguard poor operative risks; (2) to assist in diagnosis; (3) to aid in the treatment of post-operative and post-partum complications.

The Obstetrical Department has established a pre- and post-natal clinic, which is held three forenoons a week for the benefit of prospective mothers and for mothers and babies after delivery. Statistics have shown that by this means any serious complications and severe emergency operations and post partum difficulties are prevented.

The Surgical Department reports the greatest gain in any year. In all, 572 operations were performed and 460 patients were treated, an increase of 117 cases over the previous year. This represents a gain of 34 per cent. Of this number 212 cases were for abdominal conditions. Of the more important abdominal work, 98 operations were for appendicitis, 18 for gall bladder disease, 16 for stomach and intestinal lesions, 57 for hernia, and 82 for gynecological diseases. There were five deaths, a mortality of 1.2 per cent.

The X-ray Department, which was closed during the war, has been reopened under the su-

pervision of Dr. Frank E. Wheatley of Boston, who has been appointed roentgenologist of the staff. With the addition of new equipment, the hospital is now able to make any kind of roentgenological examination and to demonstrate x-ray therapy. The Pathological Department has appointed to the staff Dr. F. B. Mallory, and all pathological specimens are to be examined at his laboratory in Boston and the reports incorporated into the hospital records.

During the past year there were admitted to the hospital 752 cases: 460 surgical, 279 obstetrical and 13 medical, an increase of 116, or 18.2 per cent., over the previous year. There were nine deaths in all, five surgical, one obstetrical, and three medical. The total death rate was 1.2 per cent; surgical, 1.08 per cent.; obstetrical, 0.3 per cent.; medical, 23 per cent. The report contains a complete analysis of the work of all departments of the hospital.

The Training School for Nurses of the Godard Hospital offers a course of two and one-half years of instruction and training. The school is affiliated with the Providence City Hospital, where a three months' course of instruction is given in the care of patients with infectious diseases and in pediatrics. Three nurses were graduated in 1919.

WEEK'S DEATH RATE IN BOSTON.—During the week ending June 12, 1920, the number of deaths reported was 199 against 169 last year, with a rate of 12.84 against 11.07 last year. There were 41 deaths under one year of age against 22 last year.

The number of cases of principal reportable diseases were: Diphtheria, 29; scarlet fever, 32; measles, 189; whooping cough, 47; typhoid fever, 1; tuberculosis, 66.

Included in the above were the following cases of non-residents: Diphtheria, 10; scarlet fever, 3; measles, 1; whooping cough, 4; tuberculosis, 4.

Total deaths from these diseases were: Diphtheria, 1; whooping cough, 4; tuberculosis, 14.

Included in the above were the following non-residents: Whooping cough, 1; tuberculosis, 2.

Influenza cases, 2.

ELECTION OF DR. REID HUNT.—Dr. Reid Hunt, of Harvard University, has been elected president of the United States Pharmacopeial Convention. Dr. Hunt succeeds Dr. Harvey W. Wiley.

APPOINTMENT OF DR. STANLEY H. OSBORNE.—An appointment as director of the Division of Preventable Diseases in the Connecticut State Department of Health has been accepted by Dr. Stanley H. Osborne, formerly epidemiologist of the Massachusetts State Department of Health.

MASSACHUSETTS STATE NURSES' ASSOCIATION.—The seventeenth annual meeting of the Massachusetts State Nurses' Association was held on June 7 and 8 at the Walker Building, Boston University College of Business Administration. Over eight hundred nurses, from every part of the state, attended the meeting. On the afternoon of June 8 the opening session was held by the Private Duty Nurses' League; this was followed by a meeting of the state and local Red Cross committees and the Industrial Nurses' conference. At the general meeting in the evening, Mrs. Robert L. DeNormandie spoke on "The Importance of Public Health Nursing from the Viewpoint of a Lay Woman." An address was delivered by Miss Bernice W. Billings, New England director of the Red Cross Nursing Service, and a symposium was held on the life, nursing, training school, and vision of Florence Nightingale.

REPORT OF BABY HYGIENE ASSOCIATION.—The annual report of the Baby Hygiene Association states that 109,732 visits were made by nurses to homes during the year 1919. In 13,839 cases, special attention was given to the dieting of children of pre-school age. During the past year, at the request of the Federal health authorities, a careful study has been made of the feeding possibilities of powdered milk, and the results of tests are soon to be announced. The sum of $68,038.08 was expended during the past year in carrying on the work of the Baby Hygiene Association; this sum was raised by voluntary subscription. During this year the association plans to extend greatly its preventive work. Dr. Fritz B. Talbot is president of the association, Dr. Richard M. Smith is at the head of the medical advisory committee, and Winifred Rand, R.N., is in charge of the executive work of carrying on the work of the twenty Boston stations.

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE SOCIETY.

*First Day, June 8, 1920.*

THERE were clinics and operations at the chief hospitals of Boston during the morning, operations and demonstrations being advertised to be given at the Massachusetts Homeopathic Hospital for the first time in the history of the Society. All of the exercises of the anniversary were held at the Boston Medical Library where Fellows had an opportunity to inspect the original charter of 1781, the seal of 1783, the first record book and other early possessions besides recent volumes published by members of the Society. The exercises began with the annual meeting of the Supervisors in John Ware Hall at 11.30 A.M., followed by the meeting of the Council at noon, 130 councilors being present. The meetings of the Sections of Medicine, Surgery and Tuberculosis were held during the afternoon in the various halls of the building according to a detailed and corrected program published in the official organ of the Society, the BOSTON MEDICAL AND SURGICAL JOURNAL OF June 3, 1920, Vol. CLXXXII, pages 591-594.

The attendance and the officers elected by the five Sections for the ensuing year were as follows:

Section of Medicine: Attendance 115. *Chairman,* Herman T. Baldwin, Chestnut Hill; *Secretary,* Francis M. Rackemann, Boston.

Section of Surgery: Attendance, 135. *Chairman,* Joshua C. Hubbard, Boston; *Secretary,* George A. Leland, Jr., Roxbury.

Section of Tuberculosis: Attendance, 50. *Chairman,* E. O. Otis, Boston; *Secretary,* Sumner H. Remich, New Bedford.

Section of Hospital Administration: Attendance, 90. *Chairman,* Joseph B. Howland, Boston; *Secretary,* Nathaniel W. Faxon, Stoughton.

Section of Diseases of Children: Attendance, 180. *Chairman,* Maynard Ladd, Boston; *Secretary,* J. Herbert Young, Newton.

The Shattuck Lecture was delivered in John Ware Hall in the evening to an audience of about 200 by Dr. Allan J. McLaughlin, Assistant Surgeon General, United States Public Health Service, Washington, D. C.

Following the lecture light refreshments were served in the supper room.

*Second Day, June 9, 1920.*

The Society met at the Boston Medical Library for the exercises of the one hundred and thirty-ninth anniversary. The President, Dr. Alfred Worcester was in the chair, and one hundred and twenty-five persons were present during the morning. The reading of the minutes of the last meeting was dispensed with by vote and the minutes were adopted as published.

The Secretary stated that during the year there had been recorded 57 deaths, 35 resignations, 13 deprivations of fellowship for nonpayment of dues, and one expulsion, making a total loss for the year of 106. During this time the Council had restored 12 deprived Fellows, the Censors had readmitted five resigned Fellows and had admitted 221 new Fellows, making a gain of 238, and a net gain of 132. This number added to the membership of the Society on June 4, 1919, made the membership on June 9, 1920, 3,822, the largest in the history of the organization.

The President introduced Dr. C. W. Bartlett of Bennington, Vermont, and Dr. S. B. Overlock of Pomfret, Connecticut, recent presidents of their respective state medical societies, and they extended greetings from the Vermont State Medical Society and the Connecticut State Medical Society.

The draft of the Revised By-Laws and Code of Ethics which had been presented to the Council February 4, 1920, by the committee on revision, and had been amended and approved at that time, was brought before the Society by the President. A copy had been sent to every Fellow with the official program. When asked by the Chairman if there were any criticisms of the By-Laws or Code of Ethics, as sent out, there was no answer. On motion from the floor, duly seconded—there being forty present at the time—it was voted unanimously, That the By-Laws and Code of Ethics as revised and approved by the Council, February 4, 1920, be adopted; also Voted, That all By-Laws and the Code of Ethics heretofore in force, be and they are repealed hereby.

There being no incidental business the papers were read according to the program. At 12 o'clock noon the Annual Discourse was delivered by Dr. Hugh Cabot, Professor of Surgery at the University of Michigan. Subject: Compulsory Health Insurance, State Medicine or

What? The thanks of the Society were given to the orator by vote.

In the afternoon the Sections of Hospital Administration and Diseases of Children held their meetings according to the programs.

The annual dinner was served at the American House, Boston, to 330 Fellows and guests at 6.30 o'clock P.M. The Rev. Francis E. Webster of Waltham asked the blessing. Governor Calvin Coolidge of Massachusetts made an acceptable address and Dr. Hugh Cabot described the working of the State University in Michigan and suggested that in states such as Massachusetts, where there is no state university, it might be possible to work out a system of community medical service under the auspices of the state medical society. At nine o'clock a play in six scenes was given by the entertainment committee of the Norfolk District Medical Society entitled, "Breaking into the Army or It's a Great Life if You Don't Weaken."

Adjourned at 10.15 P.M.

WALTER L. BURRAGE,
*Secretary.*

ADMISSIONS REPORTED FROM JUNE 4, 1919, TO JUNE 9, 1920.

| Year of Admission. | Name. | Residence. | Medical College. |
|---|---|---|---|
| 1919 | Adams, Winthrop, | Cambridge | 12 |
| 1920 | Alden, Carmi Rupert, | Boston | 12 |
| 1920 | Ames, Forrest Bertram, | Dorchester | 11 |
| 1919 | Angier, Harlan Wesley, | Gilbertville | 12 |
| 1919 | Ash, Richard Maurice, | Quincy | 12 |
| 1919 | Ashmore, Buell Leslie, | Monson | 39 |
| 1920 | Atkinson, Frederick Charles, | North Andover | 12 |
| 1919 | Atkinson, Gordon Douglas, | Melrose Highlands | 22 |
| 1919 | Aub, Joseph Charles, | Boston | 11 |
| 1920 | Babcock, Harold Lester, | Boston | 10 |
| 1920 | Bachman, George Warren, | Boston | 11 |
| 1919 | Banquer, Jacob Ellis, | Mattapan | 12 |
| 1919 | Barnard, Frederick Joseph, | Worcester | 12 |
| 1920 | Belding, David Lawrence, | Hingham | 10-11 |
| 1920 | Belisle, Eugene Simeon, | Worcester | 15 |
| 1920 | Boch, Joseph, | Springfield | 11 |
| 1919 | Bock, Arlie Vernon, | Boston | 11 |
| 1920 | Bogan, Isabel Katherine, | Beverly | 33 |
| 1920 | Boland, John Joseph, | Pittsfield | 22 |
| 1919 | Brackett, Nathaniel Parker, | Worcester | 12 |
| 1920 | Breed, William Bradley, | Boston | 11 |
| 1919 | Bridgwood, David, | Brockton | 12 |
| 1920 | Briggs, Maurice Taggart, | Lynn | 11 |
| 1919 | Brown, Josiah Henry, | West Newton | 20 |
| 1919 | Brunick, Patrick Vincent, | South Boston | 12 |
| 1920 | Bunker, Harry Alden, Jr., | Medfield | 11 |
| 1920 | Burnett, Joseph Hamilton, | East Boston | 11 |
| 1920 | Burton, Oscar Augustus, | Wellesley | 45 |
| 1920 | Canedy, Frederick Snow, | Wellfleet | 10 |
| 1920 | Carey, Joseph Henry, | Dorchester | 12 |
| 1920 | Chambers, Ralph Miller, | Westborough | 16 |
| 1920 | Chapman, Willian Harden, | Hingham | 10 |
| 1919 | *Churchill, Frank Spooner, | Milton | 11 |
| 1919 | Clifford, Randall, | Boston | 11 |
| 1920 | Cline, Samuel, | Boston | 11 |
| 1919 | Clute, Howard Merrill, | Boston | 14 |
| 1919 | Cobb, Stanley, | Boston | 11 |
| 1919 | Colgate, Charles Henry, Jr., | Rockland | 10 |
| 1920 | Crawford, Joseph Warrington, | North Adams | 21 |
| 1919 | Crimmin, Leo Philip, | Brockton | 12 |
| 1919 | Cunha, Manuel Felix, | Lowell | 12 |
| 1919 | Curran, Louis Frederic, | Boston | 12 |
| 1920 | Dalrymple, Sidney Collingwood, | Newton | 5 |
| 1920 | Davidson, William Brown, | Rutland | 32-7 |
| 1920 | Dayton, Neil Avon, | Westborough | 31 |
| 1919 | Deitch, John, | Roxbury | 12 |
| 1919 | Derby, Joseph Patrick, | Worcester | 11 |
| 1920 | Dodge, Percy Loraine, | Needham | 12 |
| 1920 | Durrie, Anna Belle, | Melrose | 13 |
| 1919 | Duvally, Nicholas, | Boston | 12 |
| 1920 | Fallon, Joseph David, | Northampton | 7 |
| 1919 | Fessenden, Charles Hill, | Newton Center | 10 |
| 1920 | Finch, Edward Bronson, | Greenfield | 17 |
| 1919 | Finkelstein, Isadore Albert, | Dorchester | 12 |
| 1919 | Fipphen, Clarence Wyman, | Worcester | 11 |
| 1920 | Fisher, Edgar Alexander, | Worcester | 10 |
| 1919 | Fisher, John Charles Vincent, | Dorchester | 10 |
| 1920 | Fleury, Oswald Theodore, | Boston | 12 |
| 1919 | Foley, John Arthur, | Boston | 11 |
| 1919 | Forsley, Thomas, Jr., | Lowell | 12 |
| 1919 | Freligh, Claude Adelbert, | Palmer | 22 |
| 1920 | Friedman, Harry Falk, | Boston | 33 |
| 1920 | Frost, Harold Maurice, | Boston | 11 |
| 1919 | Fryburg, Charles August, | Worcester | 12 |
| 1920 | *Gaffney, Mary Evangeline, | Rutland | 12 |
| 1919 | Gallupe, Harold Quimby, | Boston | 11 |
| 1920 | Ganley, Edward Henry, | Methuen | 12 |
| 1919 | Garfield, Walter Thompson, | Cambridge | 11 |
| 1920 | Garland, Joseph, | Boston | 11 |
| 1919 | Gerrard, Clarence Charles, | Springfield | 22 |
| 1919 | Gillespie, Norman Wilkinson, | Dorchester | 11 |
| 1920 | Gilman, William Henry, | Cambridge | 12 |
| 1919 | Gleason, Benjamin Whitney, | Athol | 19 |
| 1919 | Godvin, Bernard Aloysius, | Jamaica Plain | 4 |
| 1919 | Goethals, Thomas Rodman, | Boston | 11 |
| 1919 | Goldman, Harry, | Roxbury | 12 |
| 1920 | Goodwin, Harold Merle, | Boston | 11 |
| 1919 | Gordon, John Hurter, | Boston | 12 |
| 1919 | Grandison, Louis Julian, | Somerville | 12 |
| 1919 | Grant, Justin Frank, | Boston | 6 |
| 1919 | Green, Hyman, | Boston | 11 |
| 1919 | Gustafson, Paul, | Boston | 11 |
| 1920 | Haggart, Gilbert Edmund, | Boston | 11 |
| 1919 | *Hamilton, Frank Andrew, | Boston | 11 |
| 1920 | Hammond, John Wilkes, Jr., | Cambridge | 11 |
| 1919 | Hanlon, Morgan Patrick, | Cambridge | 12 |
| 1920 | Harding, Edward, | Boston | 11 |
| 1920 | Hardy, Wilbert Clark, | Haverhill | 12 |
| 1920 | Harriman, Frank Edwin, | Worcester | 12 |
| 1919 | Harris, Francis Sterling, | Cambridge | 11 |
| 1919 | Harris, Walter Callahan, | Worcester | 12 |
| 1919 | Harvey, Clifford Dawes, | Brookline, | 21 |
| 1919 | Hatt, Rafe Nelson, | Newton | 12 |
| 1919 | Heffernan, Roy Joseph, | Dorchester, | 12 |
| 1919 | Herlihy, David Joseph, | Cambridge | 12 |
| 1919 | Holmes, James, | Springfield | 32 |
| 1919 | Hooper, George Henry, | Boston | 12 |
| 1920 | Hopkins, Lawrence Towle, | Belmont | 12 |
| 1919 | Howard, Perez Briggs, | Newtonville | 11 |
| 1919 | Hubbard, Eliot, Jr., | Boston | 11 |
| 1920 | Hutton, Willis Abrum, | Springfield | 24 |
| 1919 | Jackson, Arthur Morrison, | Everett | 11 |
| 1919 | Janney, James Craik, | Cambridge | 11 |
| 1919 | Jensen, William Christian, | Worcester | 12 |
| 1919 | Jordan, Michael Matthew, | Worcester | 33 |
| 1919 | Kaufman, Morris Frank, | Worcester | 12 |
| 1919 | Lancey, Clifford Scales, | Worcester | 12 |
| 1919 | Langill, Morton Howard, | Worcester | 14 |
| 1919 | Lanman, Thomas Hinckley, | Cambridge | 11 |
| 1919 | Learned, Elmer Turell, | Fall River | 11 |
| 1919 | Lena, Hugh Francis, | Lawrence | 6 |
| 1919 | Lindberg, Cosa Dell Haskell, | Quincy | 10 |

* Readmitted by Censors.

| Year of Admission. | Name. | Residence. | Medical College. |
|---|---|---|---|
| 1919 | Lindberg, | David Oscar Nathaniel, | Quincy ..10 |
| 1920 | Lipsitt, | Charles Saul, | New Bedford .....12 |
| 1920 | Loftus, | John Thomas, | Worcester .....11 |
| 1920 | Long, | Rufus Wilfred, | Boston .....12 |
| 1920 | Lunt, | Lawrence Kirby, | Concord .....11 |
| 1920 | Lurie, | Moses Hyman, | Boston .....11 |
| 1919 | Lynch, | Joseph Michael, | Dorchester .....12 |
| 1919 | MacKnight, | Richard Patton, | Fall River ....20 |
| 1920 | Macmillan, | Alexander Stewart, | West Somerville .....12 |
| 1920 | Macmillan, | Leslie Hooper, | West Somerville .12 |
| 1919 | MacPherson, | Donald John, | Boston .....11 |
| 1920 | Mann, | David Edwin, | Rutland .....12 |
| 1919 | Marnoy, | Samuel Louis, | Chelsea .....10 |
| 1919 | McDevitt, | James John, | Worcester .....30 |
| 1919 | McGuire, | Lee Wesley, | Boston .....27 |
| 1920 | Meaker, | Samuel Raynor, | Boston .....11 |
| 1920 | Meigs, | Joe Vincent, | Boston .....11 |
| 1920 | Melkonian, | Eliza Armenoohi, | Boston .....12 |
| 1919 | Meltzer, | Philip Edward, | Boston .....12 |
| 1920 | Mella, | Hugo, | Cambridge .....29 |
| 1919 | Merritt, | Edward Lester, | Fall River .....12 |
| 1920 | Mikolaitis, | Casimir John, | Lawrence .....37 |
| 1920 | Milot, | Joseph Donat, | Fall River .....38 |
| 1919 | Milward, | Francis William, Jr., | Boston .....12 |
| 1919 | Mulhern, | Joseph Patrick, | Boston .....12 |
| 1919 | Munro, | Donald, | Boston .....11 |
| 1920 | Murray, | Halstead Graeme, | Framingham ....53 |
| 1919 | Nash, | Francis Joseph, | Boston .....12 |
| 1920 | Nowell, | Howard Wilbert, | Boston .....11 |
| 1919 | Nugent, | Arthur John, | Worcester .....32 |
| 1920 | Nutter, | Denton Gove, | Newton .....11 |
| 1919 | Oberg, | Frank Thorwald, | Worcester .....11 |
| 1919 | O'Dea, | Patrick Joseph, | Fitchburg .....22 |
| 1920 | O'Hara, | Dwight, | Waltham .....11 |
| 1919 | Ormsby, | Edward Bernard, | Boston .....12 |
| 1919 | Osborn, | Stanley Hart, | Cambridge .....12 |
| 1919 | Osgood, | Howard, | Cambridge .....11 |
| 1919 | Parkhurst, | Albert Elisha, | Boston .....11 |
| 1919 | Penn, | Henry Samuel, | Lawrence .....12 |
| 1919 | Perkins, | Ralph Sherburne, | Worcester ..... 6 |
| 1920 | Perron, | Albert Ernest, | Fall River ..... 8 |
| 1919 | Pettingill, | Warren Martin, | West Somerville .12 |
| 1919 | Pfeiffer, | Albert, | Boston .....26 |
| 1919 | Pillsbury, | Nahum Roy, | Dorchester ..... 5 |
| 1919 | Plouffe, | Bernard Louis, | Webster .....11 |
| 1919 | Pobirs, | Louis Jacob, | New Bedford .....12 |
| 1919 | Porter, | William James, | Winthrop .....35 |
| 1919 | Putnam, | James Jackson, | Boston .....11 |
| 1920 | Raeder, | Oscar Jacob, | Boston ..... 3 |
| 1920 | Ragle, | Benjamin Harrison, | Brookline .....11 |
| 1920 | Redden, | William Rufus, | Boston .....11 |
| 1920 | Regan, | William Francis, | Chelsea .....12 |
| 1919 | Rice, | George Arnold, | Holden .....12 |
| 1919 | *Rice, | Herbert Augustus, | Canton .....12 |
| 1920 | Rice, | Robert, | Haverhill .....42 |
| 1920 | Richards, | Thomas Kinsman, | Boston .....11 |
| 1919 | Richardson, | Ira Walter, | Wakefield .....11 |
| 1920 | Robinson, | Bernard Herman, | Roxbury .....12 |
| 1920 | Rock, | John Charles, | Boston .....11 |
| 1919 | Rockwell, | Llewellyn Harrison, | Boston .....12 |
| 1920 | Roe, | John Cornelius, | Pittsfield ..... 4 |
| 1919 | Rosen, | Edward, | Revere .....12 |
| 1920 | Rosen, | Kermit Charles, | Dorchester .....12 |
| 1920 | Rowe, | Frank Elmer, | Revere .....22 |
| 1920 | Rowe, | Leonard Blake, | Natick .....22 |
| 1920 | Rudman, | Benjamin William, | Boston .....12 |
| 1919 | Ruggles, | Edwin Pakenham, | Dorchester .....10 |
| 1920 | Ruggles, | Ralph Hastings, | Boston .....12 |
| 1919 | Salmon, | Charles Augustus, | Worcester .....12 |
| 1920 | Sandler, | Frank Fishel, | Revere .....12 |
| 1920 | Sargent, | Arthur Forrest, | Boston ..... 11 |
| 1919 | Sargent, | Francis Barnard, | Mattapan .....11 |
| 1919 | Sartwell, | Ransom Harvey, | Foxborough ....22 |
| 1919 | Settle, | Howard Edwin, | Boston .....11 |
| 1919 | Shipton, | George Marsden, | Pittsfield ..... 6 |
| 1919 | Shortell, | Joseph Henry, | Boston .....11 |
| 1920 | Simmons, | Manfred Elliston, | Lowell .....10 |
| 1920 | Simpson, | Charles, | Southbridge .....38 |
| 1920 | Smith, | Joseph Andrew, | Worcester .....10 |
| 1919 | Solomon, | Sidney Joseph, | Everett .....12 |
| 1919 | Spellissey, | Frank Thomas, | Worcester .....12 |
| 1919 | Steinberg, | Naaman, | Boston .....12 |
| 1920 | Stickney, | Robert Cole, | Beverly .....17 |
| 1920 | Strahlmann, | Louis, | South Boston .....11 |
| 1919 | Strongman, | Bessie Talbot, | Boston ..... 6 |
| 1919 | Sullivan, | Russell Francis, | Boston .....12 |
| 1920 | Supple, | William Raymond, | Dorchester .....11 |
| 1920 | Sweeney, | John Gerard, | Hingham .....12 |
| 1919 | Taylor, | John Houghton, | Cambridge .....11 |
| 1919 | Tso, | Ernest Teh, | Boston .....11 |
| 1920 | Thorndike, | William Tecumseh Sherman, | Boston .....11 |
| 1920 | Tilton, | Warren Norwood, | Boston .....12 |
| 1920 | Tompkins, | Byron Vincent, | Sheffield .....17 |
| 1919 | Troupin, | Abraham Solomon, | Boston .....12 |
| 1920 | Ullian, | Louis Joseph, | Boston .....12 |
| 1920 | Ulrich, | Helmuth, | Weston .....10 |
| 1920 | Vaughan, | Herbert Gaines, | Attleborough ....45 |
| 1919 | Vickery, | Eugene Augustus, | Boston .....11 |
| 1919 | Viets, | Henry Rouse, Jr., | Newton .....11 |
| 1919 | *Vrooman, | Earl Morey, | North Adams ..... 8 |
| 1919 | Wagner, | Harvey Samuel, | Pocasset .....13 |
| 1920 | Waite, | Anna Jeanette, | Worcester .....33 |
| 1920 | Walker, | Edmund Eugene Watlington, | Boston 24 |
| 1919 | Watkins, | Harvey Middleton, | Monson .....39 |
| 1919 | Webster, | Frederick Alonzo, | Boston .....10 |
| 1920 | Wells, | Elwin Harrison, | Wakefield .....12 |
| 1920 | White, | Joseph Warren, | Roxbury .....11 |
| 1920 | Whitehouse, | Eugene Dizer, | Quincy .....11 |
| 1920 | Wilder, | Edward Wheeler, | Dorchester .....11 |
| 1920 | Winsor, | Allen Pellington, | Boston .....11 |
| 1919 | Wood, | William Franklin, | Boston .....12 |
| 1920 | Woodbury, | Stillman Philetus, | Millers Falls . 9 |
| 1919 | Woody, | MacIver, | Boston .....11 |
| 1919 | Wright, | Arthur Clarendon, | Haverhill .....12 |

221+5=226

* Readmitted by Censors.

KEY TO MEDICAL COLLEGES.

3 Washington University Medical School, St. Louis.
4 Georgetown University School of Medicine.
5 Bowdoin Medical School.
6 Johns Hopkins University, Medical Department.
7 College of Physicians and Surgeons, Baltimore.
8 Baltimore Medical College.
9 Long Island College Hospital, Brooklyn.
10 Boston University School of Medicine.
11 Harvard University Medical School.
12 Tufts College Medical School.
13 American Medical Missionary College.
14 Dartmouth Medical School.
15 University of Montpellier, France.
16 Cleveland Pulte Medical College.
17 Columbia University College of Physicians and Surgeons.
19 University of Pennsylvania, Dept. of Medicine.
20 Jefferson Medical College.
21 Hahnemann Medical College of Philadelphia.
22 University of Vermont, College of Medicine.
24 McGill University, Faculty of Medicine, Montreal.
26 Middlesex Hospital, London, England.
27 Medical College, University of Cincinnati.
29 University of Georgia, Medical Department.
30 University and Bellevue Hospital Medical College.
31 Ohio State University College of Medicine.
32 University of Maryland, School of Medicine.
33 Vanderbilt University, Med. Dept., Nashville, Tenn.
35 University of Virginia.
37 Chicago College of Medicine and Surgery.
38 Laval University, Quebec, Canada.
39 University of Louisville, Medical Department.
45 Rush Medical College.
53 Queen's University, Faculty of Medicine, Kingston, Canada.

* Readmitted by Censors.

DEATHS REPORTED FROM JUNE 4, 1919, TO JUNE 9, 1920.

| Admitted. | Name. | Place of Death. | Date of Death. | Age. |
|---|---|---|---|---|
| 1880 | Adams, George Edwin | Worcester | Nov. 29, 1919 | 61 |
| 1892 | †Allen, Carl Addison | Holyoke | Sept. 11, 1919 | 71 |
| 1914 | Barney, Willis Oliver | Boston | June 25, 1919 | 30 |
| 1898 | Benner, Herbert Orray | Roxbury | Dec. 22, 1919 | 54 |
| 1916 | Berr, Alfred William | Ft. Slocum, N. Y. | Oct. 8, 1918 | 30 |
| 1910 | Breen, James Henry | Hudson | Nov. 3, 1919 | 41 |
| 1894 | Brown, Daniel Joseph | Springfield | July 8, 1919 | 58 |
| 1869 | †Brown, Marshall Lebanon | Flatbush, L. I. | May 5, 1920 | 83 |
| 1897 | Cavanaugh, Charles Russell | Dorchester | December, 1919 | 50 |
| 1900 | Chase, Edwin Llewellyn | Shrewsbury | April 30, 1920 | 49 |
| 1895 | Church, Charles Albert | Millbury | Jan. 13, 1920 | 60 |
| 1896 | Clark, Joseph Eddy | Utica, N. Y. | March 4, 1920 | 62 |
| 1880 | †Clark, Julius Stimpson | Melrose | Jan. 27, 1920 | 81 |
| 1879 | Cook, Charles Henry | Natick | Dec. 3, 1919 | 74 |
| 1872 | †Cowles, Edward | Plymouth | July 25, 1919 | 82 |
| 1887 | Culbertson, Emma Valeria Pintard Bicknell | St. Petersburg, Fla. | Jan. 8, 1920 | 65 |
| 1887 | Deane, Wallace Harlow | Springfield | April 14, 1920 | 67 |
| 1861 | De Blois, Thomas Amory | Boston | Feb. 27, 1920 | 72 |
| 1891 | Frasier, Sarah Adams Bond | Jamaica Plain | June 28, 1919 | 60 |
| 1887 | Frye, Edmund Bailey | Boston | Oct. 23, 1919 | 68 |
| 1886 | Gifford, John Henry | Fall River | Dec. 14, 1919 | 61 |
| 1887 | Gilman, Warren Randall | Worcester | May 2, 1920 | 58 |
| 1861 | †Goodwin, Richard James Plumer | Malden | April 19, 1920 | 82 |
| 1871 | †Greenleaf, John Ruggles | Gardner | June 6, 1919 | 78 |
| 1914 | Houghton, Neidhard Hahnemann | Brookline | Dec. 26, 1919 | 58 |
| 1859 | †Jordan, Charles | Wakefield | June 6, 1920 | 91 |
| 1911 | Jordan, Ernest Major | Boston | March 13, 1920 | 48 |
| 1877 | Kilby, Henry Sherman | North Attleborough | April 10, 1920 | 68 |
| 1881 | Knapp, Philip Coombs | Boston | Feb. 23, 1920 | 61 |
| 1903 | Knowlton, Wallace Miles | Boston | Feb. 6, 1920 | 61 |
| 1900 | Leslie, Herbert Granville | Newburyport | Sept. 1, 1919 | 48 |
| 1879 | †Makechnie, Horace Perkins | West Somerville | Oct. 16, 1919 | 78 |
| 1869 | †Mansfield, Henry Tucker | Needham | July 6, 1919 | 81 |
| 1867 | †Millard, Henry James | North Adams | May 30, 1920 | 84 |
| 1872 | †Moran, John Brennan | Allston | July 12, 1919 | 80 |
| 1884 | Murphy, Francis Charles | Roxbury | Nov. 2, 1919 | 60 |
| 1865 | †Norris, Albert Lane | Brookline | Aug. 29, 1919 | 80 |
| 1881 | Noyes, Ernest Henry | Newburyport | Feb. 7, 1920 | 66 |
| 1855 | †Oliver, Henry Kemble | Boston | Oct. 25, 1919 | 89 |
| 1899 | Overlock, Melvin George | Worcester | Jan. 30, 1920 | 54 |
| 1882 | Palmer, Lewis Merritt | Framingham | June 4, 1919 | 68 |
| 1887 | Poirier, Emile | Salem | April 29, 1920 | 64 |
| 1867 | †Prescott, Charles Dudley | New Bedford | March 26, 1920 | 75 |
| 1865 | †Rice, Austin Bradford | Fiskdale | 1919 | 86 |
| 1883 | †Richards, George Edwards | Boston | Sept. 8, 1919 | 78 |
| 1868 | †Ruddick, William Henderson | South Boston | April 8, 1920 | 75 |
| 1906 | Russell, Frederick James | New York City | Dec. 21, 1919 | 46 |
| 1906 | Smith, Stafford Baker | Washington, D. C. | Feb. 29, 1920 | 36 |
| 1904 | Southard, Elmer Ernest | New York City | Feb. 8, 1920 | 43 |
| 1895 | Stowell, Joab | North Amherst | March 26, 1920 | 57 |
| 1863 | Swasey, Oscar Fitzallan | Beverly | June 4, 1919 | 92 |
| 1916 | Tate, Harry John | Pittsfield | Feb. 11, 1920 | 30 |
| 1855 | Twitchell, Edward Thayer | Santa Barbara, Calif. | April 6, 1920 | 57 |
| 1858 | †Underwood, George Latham | Belmont | April 1, 1920 | 88 |
| 1882 | Whitney, Edward Melville | New Bedford | Feb. 27, 1920 | 64 |
| 1903 | Yeaton, George William | Medway | Sept. 27, 1918 | 39 |
| 1913 | Zimmerman, Henry | Springfield | Aug. 7, 1919 | 30 |

Total, 57 Deaths.

† Retired Fellow

## OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY.

Chosen by the Council, June 8, 1920.

Alfred Worcester, Waltham, President.
Frederick Ellis Jones, Quincy, Vice-President.
Walter L. Burrage, Jamaica Plain, Secretary.
Arthur K. Stone, Framingham Center, Treasurer.
Edwin H. Brigham, Brookline, Librarian.

### STANDING COMMITTEES.

Of Arrangements.—C. H. Lawrence, Jr., Donald Macomber, A. W. Reggio, J. B. Swift, K. G. Percy, F. J. Callanan.

On Publications and Scientific Papers.—E. W. Taylor, R. B. Osgood, F. T. Lord, R. M. Green, A. C. Getchell.

On Ethics and Discipline.—J. W. Bartol, Henry Jackson, T. J. Robinson, David Cheever, F. W. Anthony.

On Membership and Finance.—S. B. Woodward, Algernon Coolidge, Jr., Samuel Crowell, Gilman Osgood, Homer Gage.

On Medical Education and Medical Diplomas.—Channing Frothingham, C. F. Painter, J. F. Burnham, A. G. Howard, R. L. DeNormandie.

On State and National Legislation.—Alfred Worcester, F. G. Wheatley, E. H. Stevens, F. E. Jones, J. S. Stone.

On Public Health.—E. H. Bigelow, Annie L. Hamilton, E. F. Cody, Victor Safford, R. I. Lee.

## PRESIDENTS OF DISTRICT MEDICAL SOCIETIES.

### Vice-Presidents (*Ex-officiis*).

Arranged according to seniority of fellowship in The Massachusetts Medical Society.

F. B. Lund ..... Suffolk
F. H. Baker ..... Worcester
S. A. Clark ..... Hampshire
C. L. Sopher ..... Middlesex East
E. J. Welch ..... Middlesex North
H. T. Baldwin ..... Middlesex South
F. J. Hanley ..... Plymouth
C. E. Woods ..... Worcester North
E. H. Bushnell ..... Norfolk South
D. D. Murphy ..... Essex North
W. G. Phippen ..... Essex South
H. G. Ripley ..... Bristol North
G. W. Winchester ..... Norfolk
C. P. Curley ..... Barnstable
A. P. Merrill ..... Berkshire
J. A. Mather ..... Franklin
J. M. Birnie ..... Hampden
E. D. Gardner ..... Bristol South

## COUNCILORS, 1920-1921.

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, APRIL 15 TO MAY 15, 1920.

NOTE.—The initials M. N. C., following the name of a councilor, indicate that he is a member of the Nominating Committee. V.-P. indicates that a member is a councilor by virtue of his office as president of a district society, and so vice-president of the general society. C. indicates that he is chairman of a Standing Committee. Ex-P. indicates ex-President.

BARNSTABLE,
C. P. Curley, V.P., Provincetown.
W. D. Kinney, Osterville.
J. P. Nickerson, M.N.C., West Harwich.
E. F. Curry, Sagamore.

BERKSHIRE,
A. P. Merrill, V.P., Pittsfield.
Henry Colt, Pittsfield.
B. W. Paddock, Pittsfield.
W. L. Tracy, M.N.C., Pittsfield.
P. J. Sullivan, Dalton.

BRISTOL NORTH,
H. G. Ripley, V.P., Taunton.
W. H. Allen, Mansfield.
W. O. Hewitt, Attleborough.
F. A. Hubbard, M.N.C., Taunton.

BRISTOL SOUTH,
E. D. Gardner, V.P., New Bedford.
E. F. Cody, New Bedford.
C. F. Connor, M.N.C., New Bedford.
A. B. Cushman, South Dartmouth.
W. A. Dolan, Fall River.
R. W. Jackson, Fall River.
A. C. Lewis, Fall River.
A. H. Mandell, New Bedford.

ESSEX NORTH,
D. D. Murphy, V.P., Amesbury.
R. V. Baketel, M.N.C., Methuen.
J. F Burnham, Lawrence.
I. J. Clarke, Haverhill.
T. R. Healy, Newburyport.
G. E. Kurth, Lawrence.
F. D. McAllister, Lawrence.
F. B. Pierce, Haverhill.
F. E. Sweetsir, Merrimac.

ESSEX SOUTH,
W. G. Phippen, V.P., Salem.
H. P. Bennett, Lynn.
R. E. Bicknell, Lynn.

ESSEX SOUTH—*continued*.
S. P. F. Cook, Gloucester.
J. F. Donaldson, Salem.
S. C. Eveleth, Marblehead.
H. K. Foster, Peabody.
W. T. Hopkins, M.N.C., Lynn.
J. F. Jordan, Peabody.
G. M. Kline, Beverly.
S. W. Mooring, Gloucester.
A. N. Sargent, Salem.
R. E. Stone, Beverly.

FRANKLIN,
J. A. Mather, V.P., Greenfield.
G. P. Twitchell, M.N.C., Greenfield.
C. L. Upton, Shelburne Falls.

HAMPDEN,
J. M. Birnie, V.P., Springfield.
F. H. Allen, Holyoke.
E. P. Bagg, Jr., Holyoke.
R. S. Benner, Springfield.
H. F. Byrnes, Springfield.
J. J. Carroll, Holyoke.
A. C. Eastman, Springfield.
M. B. Hodskins, Monson.
G. H. Janes, Westfield.
Philip Kilroy, M.N.C., Springfield.
E. A. Knowlton, Holyoke.
L. E. Mannix, Chicopee.
H. C. Martin, Springfield.
G. L. Schadt, Springfield.

HAMPSHIRE,
S. A. Clark, V.P., Northampton.
J. G. Hanson, Northampton.
C. E. Perry, Haydenville.
D. M. Ryan, M.N.C., Ware.

MIDDLESEX EAST,
C. L. Sopher, V.P., Wakefield.
D. T. Buzzell, Wilmington.
C. E. Ordway, Winchester.
R. D. Perley, M.N.C., Melrose.
P. H. Provandie, Melrose.

MIDDLESEX NORTH,
E. J. Welch, V.P., Lowell.
W. B. Jackson, Lowell.
J. H. Lambert, Lowell.
G. A. Leahey, Lowell.
J. A. Mehan, Lowell.
J. H. Nichols, M.N.C., Tewksbury.
M. A. Tighe, Lowell.

MIDDLESEX SOUTH,
H. T. Baldwin, V.P., Chestnut Hill.
D. C. Dow, Cambridge.
A. W. Dudley, Cambridge.
F. J. Goodridge, Cambridge.
L. S. Hapgood, Cambridge.
F. R. Jouett, Cambridge.
E. H. Stevens, M.N.C., Cambridge.
H. P. Walcott, Ex-P., Cambridge.
John Duff, Charlestown.
H. J. Keaney, Everett.
Godfrey Ryder, Malden.
C. H. Staples, Malden.
C. E. Mongan, Somerville.
W. D. Ruston, West Somerville.
L. F. Sise, Medford.
G. W. W. Whiting, Somerville.
C. O. Chase, Watertown.
Richard Collins, Waltham.
C. A. Dennett, Arlington.
W. E. Fernald, Waverley.
Alfred Worcester, Pres., Waltham.
E. A. Andrews, Newton.
F. G. Curtis, Chestnut Hill.
H. S. Rowen, Brighton.
F. R. Stubbs, Newton.
G. R. West, Newton Center.
G. W. Gay, Ex-P., Chestnut Hill.
C. E. Hills, South Natick.

MIDDLESEX SOUTH—*continued.*
G. A. Bancroft, Natick.
E. H. Bigelow, C., Framingham Center.
A. K. Stone, Treas., Framingham Center.
Fresenius Van Nüys, Weston.

NORFOLK,
G. W. Winchester, V.P., Mattapan.
C. E. Allard, Dorchester.
W. B. Batchelder, Dorchester.
D. N. Blakely, Brookline.
E. J. Brearton, Dorchester.
E. H. Brigham, Libra., Brookline.
F. P. Broderick, Jamaica Plain.
A. N. Broughton, M.N.C., Jamaica Plain.
W. L. Burrage, Sec'y, Jamaica Plain.
W. H. Greene, Roxbury.
W. A. Griffin, Sharon.
R. W. Hastings, Brookline.
H. T. Holland, Jamaica Plain.
G. W. Kaan, Brookline.
W. B. Keeler, Roxbury.
Bradford Kent, Dorchester.
F. P. McCarthy, Milton.
A. P. Perry, Jamaica Plain.
M. V. Pierce, Milton.
H. H. Powers, Brookline.
Victor Safford, Jamaica Plain.
G. H. Scott, Roxbury.
Max Sturnick, Roxbury.
W. J. Walton, Dorchester.
Augusta G. Williams, Brookline.

NORFOLK SOUTH,
E. H. Bushnell, V.P., Quincy.
C. S. Adams, Wollaston.
F. E. Jones, Vice.-Pres., Quincy.
J. H. Libby, East Weymouth.
D. B. Reardon, Quincy.
G. H. Ryder, M.N.C., Quincy.

PLYMOUTH,
F. J. Hanley, V.P., Whitman.
W. C. Keith, Brockton.
C. E. Lovell, Whitman.
Gilman Osgood, Rockland.
F. J. Ripley, Brockton.
F. G. Wheatley, M.N.C., North Abington.

SUFFOLK,
F. B. Lund, V.P., Boston.
J. L. Ames, Boston.
S. H. Ayer, Boston.
J. W. Bartol, C., Boston.
Robert Bonney, East Boston.
J. T. Bottomley, Boston.
V. Y. Bowditch, Boston.
E. G. Brackett, Boston.
J. E. Briggs, Boston.
F. J. Cotton, Boston.
E. A. Crockett, Boston.
Loretta J. Cummins, Boston.
Lincoln Davis, Boston.
Channing Frothingham, C., Boston.
W. J. Gallivan, South Boston.
J. E. Goldthwait, Boston.
G. S. Hill, Boston.
W. C. Howe, M.N.C., Boston.
F. L. Jack, Boston.
Henry Jackson, Boston.
D. F. Jones, Boston.
F. A. Locke, Boston.
F. T. Lord, Boston.
R. H. Miller, C., Boston.
J. J. Minot, Boston.
F. S. Newell, Boston.
E. H. Nichols, Boston.
B. W. Pond, Boston.
Edward Reynolds, Boston.
W. H. Robey, Jr., Boston.
Stephen Rushmore, Boston.
D. D. Scannell, Boston.
C. L. Scudder, Boston.

SUFFOLK—*continued.*
G. B. Shattuck, Ex-P., Boston.
C. M. Smith, Boston.
Myles Standish, Boston.
J. S. Stone, Boston.
E. W. Taylor, C., Boston.
Louisa P. Tingley, Boston.
F. H. Williams, Boston.

WORCESTER,
F. H. Baker, V.P., Worcester.
W. P. Bowers, Ex-P., Clinton.
W. J. Delahanty, Worcester.
G. E. Emery, Worcester.
M. F. Fallon, Worcester.
Homer Gage, Worcester.
J. O. Généreux, Webster.
R. W. Greene, Worcester.
David Harrower, M.N.C., Worcester.
E. L. Hunt, Worcester.
A. G. Hurd, Millbury.
W. L. Johnson, Uxbridge.
G. F. O'Day, Worcester.
C. B. Stevens, Worcester.
G. O. Ward, Worcester.
F. H. Washburn, Holden.
C. D. Wheeler, Worcester.
S. B. Woodward, Ex-P., C., Worcester.

WORCESTER NORTH,
C. E. Woods, V.P., Lunenburg.
W. E. Currier, Leominster.
E. L. Fiske, M.N.C., Fitchburg.
J. G. Henry, Winchendon.
W. F. Sawyer, Fitchburg.

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## CENSORS, 1920-1921.

BARNSTABLE,
J. P. Nickerson, Supervisor, West Harwich.
C. E. Harris, Hyannis.
E. E. Hawes, Hyannis.
J. H. Higgins, Marstons Mills.
E. S. Osborne, West Dennis.

BERKSHIRE,
Henry Colt, Supervisor, Pittsfield.
A. C. England, Pittsfield.
J. F. Crowley, Adams.
O. J. Brown, North Adams.
E. H. Howard, Pittsfield.

BRISTOL NORTH,
F. A. Hubbard, Supervisor, Taunton.
H. B. Baker, Taunton.
T. F. Clark, Taunton.
A. R. Crandell, Taunton.
T. J. Robinson, Taunton.

BRISTOL SOUTH,
W. A. Dolan, Supervisor, Fall River.
W. E. Blaine, Mattapoisett.
W. T. Learned, Fall River.
C. J. Leary, New Bedford.
S. V. Merritt, Fall River.

ESSEX NORTH,
F. D. McAllister, Supervisor, Lawrence.
J. E. Bryant, Haverhill.
R. C. Hurd, Newburyport.
A. M. Hubbell, Haverhill.
F. S. Smith, North Andover.

ESSEX SOUTH,
J. F. Donaldson, Supervisor, Salem.
A. T. Hawes, Lynn.
F. W. Baldwin, Danvers.
J. A. Shatswell, Beverly.
J. W. Trask, Lynn.

FRANKLIN,
C. L. Upton, Supervisor, Shelburne Falls.
H. N. Howe, Greenfield.
R. H. Philbrick, Northfield.
A. L. Newton, Northfield.
Charles Moline, Sunderland.

HAMPDEN,
R. S. Benner, Supervisor, Springfield.
J. M. Tracy, Springfield.
M. W. Harrington, Indian Orchard.
O. R. Blair, Springfield.
E. L. Davis, Springfield.

HAMPSHIRE,
J. G. Hanson, Supervisor, Northampton.
J. E. Hayes, Northampton.
N. C. Haskell, Amherst.
W. J. Collins, Northampton.
W. B. Segur, Enfield.

MIDDLESEX EAST,
P. H. Provandie, Supervisor, Melrose Highlands.
I. T. Cutter, Winchester.
T. E. Caulfield, Woburn.
C. R. Henderson, Reading.
J. P. Bixby, Woburn.

MIDDLESEX NORTH,
J. H. Lambert, Supervisor, Lowell.
E. J. Clark, Lowell.
G. O. Lavalée, Lowell.
J. A. Gage, Tyngsboro.
J. P. McAdams, Lowell.

MIDDLESEX SOUTH,
F. J. Goodridge, Supervisor, Cambridge.
F. G. Smith, Somerville.
C. B. Fuller, Waltham.
I. J. Fisher, West Newton.
James Glass, Framingham.

NORFOLK,
W. J. Walton, Supervisor, Dorchester.
B. S. Blanchard, Brookline.
C. F. Stack, Hyde Park.
E. T. Rollins, Jamaica Plain.
T. J. Murphy, Roxbury.

NORFOLK SOUTH,
J. H. Libby, Supervisor, East Weymouth.
W. J. Middleton, Quincy.
F. R. Dame, Braintree.
W. L. Sargent, Quincy.
V. M. Tirrell, South Weymouth.

PLYMOUTH,
F. J. Ripley, Supervisor, Brockton.
J. H. Drohan, Brockton.
E. J. Beaulieu, Whitman.
W. W. Fullerton, Brockton.
L. B. Reed, Plymouth.

SUFFOLK,
C. M. Smith, Supervisor, Boston.
T. J. O'Brien, Boston.
J. S. Stone, Boston.
R. H. Vose, Boston.
G. B. Fenwick, Chelsea.

WORCESTER,
F. H. Washburn, Supervisor, Holden.
W. H. Rose, Worcester.
E. H. Mackay, Clinton.
C. A. Sparrow, Worcester.
A. E. O'Connell, Worcester.

WORCESTER NORTH,
W. F. Sawyer, Supervisor, Fitchburg.
G. P. Norton, Fitchburg.
H. R. Nye, Leominster.
C. H. Jennings, Fitchburg.
C. S. Brigham, Leominster.

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## COMMISSIONERS OF TRIALS.

### 1920-1921.

BARNSTABLE, F. A. Binford, Hyannis.
BERKSHIRE, F. C. Downing, Stockbridge.
BRISTOL NORTH, C. S. Holden, Attleborough.
BRISTOL SOUTH, P. T. Crispo, Fall River.
ESSEX NORTH, J. F. Croston, Haverhill.
ESSEX SOUTH, J. E. Simpson, Salem.
FRANKLIN, F. E. Johnson, Irving.
HAMPDEN, E. A. Bates, Springfield.
HAMPSHIRE, W. H. Adams, Northampton.
MIDDLESEX EAST, V. C. Stewart, Woburn.
MIDDLESEX NORTH, F. E. Varney, North Chelmsford.
MIDDLESEX SOUTH, A. G. Griffin, Malden.
NORFOLK, M. V. Pierce, Milton.
NORFOLK SOUTH, N. S. Hunting, Quincy.
PLYMOUTH, Gilman Osgood, Rockland.
SUFFOLK, Channing Frothingham, Boston.
WORCESTER, W. P. Bowers, Clinton.
WORCESTER NORTH, E. P. Miller, Fitchburg.

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## OFFICERS OF THE DISTRICT MEDICAL SOCIETIES, 1920-1921.

Elected by the District Medical Societies between April 15 and May 15.

BARNSTABLE.—C. P. Curley, Provincetown, *President;* P. F. Miller, Harwich, *Vice-President;* C. J. Bell, Wellfleet, *Secretary;* H. B. Hart, Yarmouthport, *Treasurer;* E. E. Hawes, Hyannis, *Librarian.*

BERKSHIRE.—A. P. Merrill, Pittsfield, *President;* G. P. Hunt, Pittsfield, *Vice-President;* O. L. Bartlett, Pittsfield, *Secretary;* C. T. Leslie, Pittsfield, *Treasurer.*

BRISTOL NORTH.—H. G. Ripley, Taunton, *President;* Sumner Coolidge, Middleborough, *Vice-President;* A. R. Crandell, Taunton, *Secretary;* R. D. Dean, Taunton, *Treasurer.*

BRISTOL SOUTH.—E. D. Gardner, New Bedford, *President;* A. I. Connell, Fall River, *Vice-President;* A. J. Abbe, Fall River, *Secretary and Treasurer.*

ESSEX NORTH.—D. D. Murphy, Amesbury, *President;* F. W. Snow, Newburyport, *Vice-President;* J. F. Burnham, Lawrence, *Secretary and Treasurer.*

ESSEX SOUTH.—W. G. Phippen, Salem, *President;* Loring Grimes, Swampscott, *Vice-President;* G. E. Tucker, Salem, *Secretary;* G. Z. Goodell, Salem, *Treasurer;* C. M. Cobb, Lynn, *Librarian.*

FRANKLIN.—J. A. Mather, Greenfield, *President;* E. C. Thorn, Deerfield, *Vice-President;* F. A. Millett, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—J. M. Birnie, Springfield, *President;* M. B. Hodskins, Monson, *Vice-President;* H. L. Smith, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—S. A. Cook, Northampton, *President;* A. J. Bonneville, Hatfield, *Vice-President;* E. E. Thomas, Northampton, *Secretary and Treasurer;* F. E. Dow, Northampton, *Librarian.*

MIDDLESEX EAST.—C. L. Sopher, Wakefield, *President;* G. F. Dow, Reading, *Vice-President;* A. E. Small, Melrose, *Secretary;* Richard Dutton, Wakefield, *Treasurer;* G. W. Nickerson, Stoneham, *Librarian.*

MIDDLESEX NORTH.—E. J. Welch, Lowell, *President;* W. M. Jones, Lowell, *Vice-President;* J. Y. Rodger, Lowell, *Secretary;* T. B. Smith, Lowell, *Treasurer;* P. J. Meehan, Lowell, *Librarian.*

MIDDLESEX SOUTH.—H. T. Baldwin, Chestnut Hill, *President;* E. H. Bigelow, Framingham Centre, *Vice-President;* F. B. M. Cady, Cambridge, *Secretary;* Edward Mellus, Newton, *Treasurer.*

NORFOLK.—G. W. Winchester, Mattapan, *President;* C. D. Knowlton, Roxbury, *Vice-President;* Bradford Kent, Dorchester, *Secretary;* G. W. Kaan, Brookline, *Treasurer.*

NORFOLK SOUTH.—E. H. Bushnell, Quincy, *President;* F. E. Jones, Quincy, *Vice-President;* C. A. Sullivan, South Braintree, *Secretary, Treasurer, and Librarian.*

PLYMOUTH.—F. J. Hanley, Whitman, *President;* R. B. Rand, North Abington, *Vice-President;* W. C. Keith, Brockton, *Secretary, Treasurer, and Librarian.*

SUFFOLK.—F. B. Lund, Boston, *President;* J. W.

Bartol, Boston, *Vice-President;* R. H. Miller, Boston, *Secretary;* D. J. Bristol, Jr., Boston, *Treasurer;* W. P. Coues, Boston, *Librarian.*

WORCESTER.—F. H. Baker, Worcester, *President;* J. J. Goodwin, Clinton, *Vice-President;* G. A. Dix, Worcester, *Secretary;* G. O. Ward, Worcester, *Treasurer;* A. C. Getchell, Worcester, *Librarian.*

WORCESTER NORTH.—C. E. Woods, Lunenburg, *President;* R. S. Ely, West Townsend, *Vice-President;* C. H. Jennings, Fitchburg, *Secretary;* F. H. Thompson, Jr., Fitchburg, *Treasurer;* L. F. Baker, Fitchburg, *Librarian.*

## Miscellany.

### PNEUMONIA AND FATIGUE.

IN the issue of the JOURNAL for Oct. 31, 1918, was published an article by Dr. William N. Cowles of Boston on "Fatigue as a Contributory Cause of Pneumonia." This paper received some comment in the daily press at the time and was followed by the publication of a letter on the same subject by Dr. Samuel Delano, of this city, which seems so pertinent to the matter as to deserve appearance in these columns:

As a physician who has bestowed some little thought upon exercise and, in book form, given recent expression to his views, I am grateful to the *Herald* for its editorial on "Pneumonia and Fatigue." I find it a very important topic. Early in the war I said that I hoped we should learn how much stamina there was back of athletics. Muscle is more or less of a trick and the capacity to perform an athletic stunt can always be acquired through sufficient application; but the specific feat takes account of the body only as a machine; which is a distinctly opprobrious term. For the body is a vital machine, and our test of its perfection the possession of vitality, by which we mean the power to last and of stamina, which is another way of saying—power to resist.

The gathering together of so many men with all manner of previous training, and with no inconsiderable body of them previously hard drivers in athletics, would seem to offer a golden opportunity for studying the relation between a man's antecedent habits and his capacity to ward off sickness and disease. I fear me, though, the occasion will not find the man. He would have to be a medical practitioner of broad experience and logical mind, and individual cases would have to be studied carefully. Still, should there prove to be a glaring discrepancy between forceful athletics and possession of vital force, the deduction might be made in a broadly general way.

Out of our experience with the present epidemic, one is forced to the preliminary conclusion, however, that in the cantonments stamina must be minus or vital powers at a low ebb, because, taking the community at large, the mortality has been anything but uniform; from that point of view the cantonment figures mount to a height far and away beyond any proportionate average.

The training program of the cantonments, both on paper and from what we can hear, has seemed appalling. It appears to rest on the principle that the more you take out of a man the more you put in. A young officer detailed for work put to me the question whether three-quarters of an hour of setting-up directly after breakfast, followed by a run around, was reasonable. He told me he protested, and I assured him I should. The whole subject deserves keen analysis. Work is work, but exercise need not be work. Above all, a setting-up shouldn't be work at all.

It is to be feared that the athletic instructors cannot be at all relied upon to draw conclusions for guidance. They are very keen propagandists and not far-seeing. Too much of their advocacy is based upon the tenet that whatever is is right, and the argument is moved up accordingly. This scarcely meets the demands of analysis and logic. Only yesterday "Old Bill" was using athletics to account for all our success—and presently the whole world is to be doing one hundred yards in ten seconds, to its eternal advantage. One can see where the cultivation of sports is a great school for actual fighting, without being blind to the fact that actual fighting is but a relatively small part of the business of being a soldier. The desideratum is: to carry a man through the whole varied business of being a soldier without his succumbing.

SAMUEL DELANO, M.D.

### PRELIMINARY REPORT OF UNITED STATES BIRTH STATISTICS.

IN the birth-registration area of the United States 1,353,792 infants were born alive in 1917, representing a birth rate of 24.6 per 1,000 of population. The total number of deaths in the same area was 776,222, or 14.1 per 1,000. The births exceeded the deaths by 74.4 per cent. For every state in the registration area, for practically all the cities, and for nearly all the counties, the births exceeded the deaths, in most cases by considerable proportions. The mortality rate for infants under 1 year of age averaged 93.8 per 1,000 living births.

The birth-registration area, established in 1915, has grown rapidly. It comprised, in 1917, the six New England states, Indiana, Kansas, Kentucky, Maryland, Michigan, Minnesota, New York, North Carolina, Ohio, Pennsylvania, Utah, Virginia, Washington, Wisconsin, and the District of Columbia, and had an estimated population of 55,000,000, or about 53 per cent of the estimated total population of the United States in that year.

The birth rate for the entire birth-registration area fell below that for 1916 by two-tenths of one per 1,000 population; but the death rate

was less by six-tenths of one per 1,000 than in 1916. Thus the excess of the birth rate over the death rate for 1917, which amounted to 10.5 per 1,000, was somewhat greater than the corresponding excess for 1916, 10.1 per 1,000 although it fell slightly below that for 1915, 10.9 per 1,000. If the birth and death rates prevailing in any one of these three years were to remain unchanged, and if no migration were to take place to or from the area to which they relate, its population would increase at the rate of slightly more than 1 per cent per annum, or a little more than 10 per cent in a decade. This would be about half the rate—21 per cent—by which the entire population of the United States increased between 1900 and 1910.

Of the total number of births reported, 1,280,288, or 24.5 per 1,000, were of white infants, and 73,504, or 25.8 per 1,000, were of colored infants. The death rates for the two elements of the population were 13.7 and 22.5 per 1,000, respectively.

The infant mortality rate—that is, the number of deaths of infants under 1 year of age per 1,000 born alive—throughout the birth-registration area as a whole was 93.8 in 1917, as against 101 in 1916 and 100 in 1915. This is equivalent to saying that in 1915 and 1916, of every 10 infants born alive 1 died before reaching the age of 1 year, whereas in 1917 the corresponding ratio was a trifle more than 1 in 11. Among the 20 states these rates ranged from 67.4 for Minnesota to 119.9 for Maryland; and for the white population separately the lowest and the highest rates were 66.3 for Washington and 109.5 for New Hampshire.

The infant mortality rates vary greatly for the two sexes and for the various nationalities. The rate for male infants in 1917, 103.7 per 1,000 living births, was nearly 25 per cent greater than that for female infants, which was only 83.3. When the comparison is made on the basis of race or nationality of mother a minimum of 66.2 per 1,000 births is shown for infants with mothers born in Denmark, Norway and Sweden, and a maximum of 172.6 for infants with mothers born in Poland, while for Negro children the rate was 148.6.

The reports from the registration area show the birth of 14,394 pairs of twins and 155 sets of triplets in 1917—in all, 29,253 infants, or a little more than 2 per cent of the total number born.

The reports for 1,241,722 of the births occurring in 1917 contained information as to number of child in order of birth. Of these reports, 339,042 were for the first child born to the mother, 264,044 for the second child, 191,528 for the third, 134,331 for the fourth, and 95,931 for the fifth. In the remaining 216,846 cases, or 17.5 per cent of the entire number for which information upon this point was obtained, the total number of children borne by the mother was 6 or more; in 37,914 cases it was 10 or more; in 1,600 cases, 15 or more; in 56 cases, 20 or more; and in one case, that of a colored woman, the birth of a twenty-fifth child was reported.

The total number of children borne by the mothers who gave birth to these 1,241,722 infants in 1917, in whose cases data were available as to previous births, was 4,093,908. The reports for 1,194,621 of the births occurring in 1917 contained information as to the entire number of children borne by the mothers and still living, and gave a total of 3,443,466, or an average of very nearly 3 living children in each family in which a birth took place in 1917.

## A PHYSICIAN OF THE EARLY CHRISTIAN ERA.

THE *Lancet* has recently published the following item about a forgotten physician of the second century of the Christian era.

Samuel, surnamed Schabour, and also Arioch, and mentioned in Jewish literature as Mar Samuel was born at Nehardea (destroyed by Odenathus in A. D. 259) about A. D. 160. His father, Abba, was an important person there. Physically insignificant, being abnormally short and ill-developed, intellectually Samuel was great. He was educated at Nehardea by Rabbi Leir, a learned man who turned out several famed pupils. Samuel studied and specialized in various branches of learning, but his chief reputation and, indeed, the profession he finally made his own, was that of the healing art. As a student he early made such progress in medicine that he was permitted to perform autopsies, and we have records of some of these. There is an account of a decision of his as to how large a piece of skull could safely be removed in case of fracture. He is quoted as stating after examination of an aborted fœtus that it was 41 days old, showing an intimate knowledge of the conformation of the product of conception at various stages. His opinion upon the abnormality termed spina bifida is also upon record. He must have carried out anatomical dissections, because there are extant quotations from his views on the limit of the spinal marrow. He appears to have been cognisant of the lacrimal gland and of the atrophy of which it is susceptible under certain circumstances in advanced age. His treatise upon the pathogenic symptoms following upon abrupt changes of diet is quite modern in thought. All varieties of regimen, he said, are liable to be the starting point of some malady.

For external diseases he sought natural causes for their occurrence and for their fatal termination; so for penetrating sores he accused the air of rendering them incurable. Also for wounds which finally poisoned the system, he said the cause was some virus upon the weapon that had inflicted the injury. In pathology he endeavored to indicate for each malady the characteristic symptoms: for grave rhinitis, following prob-

ably a polypus ulcer, he said it could be detected by the offensive odor proceeding from the nasal organ. Migraine he attributed to excessive solitude and introspection. Magic as a curative he ignored. As to the propriety of attempting cures upon the Sabbath he was entirely favorable. He advocated the use of the speculum in order to ascertain if hemorrhage proceeded from the vagina or the uterus. He was a great believer in bleeding as a remedy for many ills. Cleanliness was a main feature of his preventive teaching. The hands should be washed at least twice a day to prevent eye affections. He admitted his inability to cure three illnesses: that proceeding from eating green dates, if unripe and sour; the evil consequences of wearing a damp linen waistband as a girdle; and illness caused from going to bed after eating too hearty a meal and taking no exercise. Perhaps he also intended to convey that patients acting so foolishly were unworthy of being given remedial relief.

## UNITED STATES CIVIL-SERVICE EXAMINATION.

### JUNIOR PHYSIOLOGIST.

July 6, 1920.

The United States Civil Service Commission announces an open competitive examination for junior physiologist. A vacancy at Edgewood Arsenal, Edgewood, Md., at $2,000 a year and quarters for single employee, and vacancies in positions requiring similar qualifications, at this or higher or lower salaries, will be filled from this examination, unless it is found in the interest of the service to fill any vacancy by reinstatement, transfer, or promotion.

Appointees whose services are satisfactory may be allowed the temporary increase granted by Congress of $20 a month. There is only a possibility of this temporary increase being allowed the appointee at Edgewood Arsenal.

In filling vacancies in positions with headquarters outside of Washington, D. C., certification will be made of the highest eligibles residing nearest the vicinity of the place at which the appointee is to be employed, except that upon the request of the department certification will be made of the highest eligibles on the register for the entire country who have expressed willingness to accept appointment where the vacancy exists.

All citizens of the United States who meet the requirements, both men and women, may enter this examination: appointing officers, however, have the legal right to specify the sex desired in requesting certification of eligibles. For the present vacancy male eligibles are desired.

The duties of the appointee will be to assist in extensive work on toxic compounds.

Competitors will not be required to report for examination at any place, but will be rated on the following subjects, which will have the relative weights indicated, on a scale of 100: (1) Physical ability, 10; (2) Training and experience, 90.

*Competitors will be rated upon the sworn statements in their applications and upon corroborative evidence.*

Applicants must have completed a four years' high-school course or have equivalent education. In addition they must have completed two years of a course in a medical school of recognized standing, qualifying in physiology, pharmacology, and pathology, with laboratory work, or have had at least one year's experience in laboratory work and experimental pharmacology and toxicology.

Applicants must have reached their twenty-first birthday on the date of the examination.

Applicants must submit with their applications their unmounted photographs, taken within two years, with their names written thereon. Proofs or group photographs will not be accepted. Photographs will not be returned to applicants.

Applicants will be admitted to this examination regardless of their residence and domicile; but only those who have been actually domiciled in the State or Territory in which they reside for at least one year previous to the examination, and who have the county officer's certificate in the application form executed, may become eligible for permanent appointment to the apportioned service in Washington, D. C.

Applicants should at once apply for Form 1312, stating the title of the examination desired, to the Civil Service Commission, Washington, D. C.: the Secretary of the United States Civil Service Board, Customhouse, Boston, Mass., New York, N. Y., New Orleans, La., Honolulu, Hawaii; Post Office, Philadelphia, Pa., Atlanta, Ga., Cincinnati, Ohio, Chicago, Ill., St. Paul, Minn., Seattle, Wash., San Francisco, Calif.; Old Customhouse, St. Louis, Mo.; Administration Building, Balboa Heights, Canal Zone; or to the Chairman of the Porto Rican Civil Service Commission, San Juan, P. R.

Applications should be properly executed, excluding the medical certificate, and must be filed with the Civil Service Commission, Washington, D. C., prior to the hour of closing business on July 6, 1920.

## Correspondence.

### PHYSICIANS IN POLITICS.

Milford, Mass., June 4, 1920.

*Mr. Editor:*

The editorial in the JOURNAL of May 13 on "Legislation on Medical Matters" is bearing fruit. We have in this section of the state a local medical society which was organized 67 years ago, and has held regular meetings ever since—which we think is doing pretty well in consideration of the sparseness of our population and the limited area to which we are confined. We are country doctors, we read the JOURNAL, and we think for ourselves.

The particular fruit to which I allude was a resolution adopted unanimously at our June meeting, yesterday. I give it without further comment.

> "*Resolved*, That the Thurber Medical Association hereby strongly endorses the candidacy of Dr. William L. Johnson of Uxbridge for Senator from the Fourth Worcester District. Important questions of public health are multiplying each year before the legislature, and men are needed to deal with these questions who understand them. These considerations, coupled with the extremely small number of medical men to be found in that body, lead us to favor Dr. Johnson for this important office. In his three years of service in the lower house, he has shown himself an efficient member, a convincing speaker, a man of convictions, who is not afraid to stand by them. Believing that men of this character are needed in the legislature, we appeal to the voters of the district to ratify our endorsement of Dr. Johnson at the polls."

We took this action not in the spirit of partisan politics, but in the interest of public health; and in this spirit we send you this note.

F. T. HARVEY, *President.*
J. M. FRENCH, *Secretary.*